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THE TREATMENT OF POLIOMYELITIS.[1]

By LAURENCE MACDONALD, M.Ch. (Orth.), F.R.C.S.,
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So much has been written and said about poliomyelitis in the last few years that I approached the preparation of this paper with considerable misgiving. I felt that I had nothing new to say, and that reiteration of well-worn themes would be quite unproductive. But a perusal of recent literature gives much food for thought; and though it would appear that no discoveries have as yet been made which offer a sure basis for preventive or curative therapy, many of the mysteries of the disease have been unravelled and we are slowly arriving at some understanding of virus infections.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on December 9, 1937.

The problem of prevention is one that has occupied the attention of public health authorities the world over.(1) In the last six years Canada, the United States of America, and Denmark have been the chief sufferers from poliomyelitis, but we in Australia have had a fairly high incidence in proportion to our population. While it is true that as a fatal disease poliomyelitis is of small account when compared with many of the other ills of childhood, the summation of its effects over a period of years has produced a large body of permanent cripples, the bulk of whom throughout life are dependent on the State for support.

I do not propose to discuss the symptoms and signs of the disease. These are now almost public property, and the recent newspaper campaign must have caused much useless anxiety to parents.

It is commonly held that infection is due to contact between a susceptible individual and one

suffering from the disease, and on experimental grounds the incubation period is said to vary from six to eighteen days. Many eminent authorities maintain that the virus dies quickly, that infectiousness is short lived, and that the chronic carrier does not exist,[2] though it is known that in monkeys experimentally infected the nasal secretion may contain active virus over a period of many weeks. In most hospitals it has been the custom to admit acute cases to the ordinary wards, but our beliefs, founded on past experience, have been rudely shaken by the present epidemic in Victoria. The problem of the carrier is still unsettled, but there is no doubt that the infective period may last for several weeks, so that isolation of the patient is of paramount importance.

One of the greatest problems in epidemiology is set by the tendency of poliomyelitis to arise sporadically in centres far apart. In Denmark, for no apparent reason, isolated villages have been attacked. These facts are difficult to explain on the contact theory of spread, and Hjalmar Wennerberg, dissatisfied with this explanation, has postulated an interesting theory of insect dissemination, probably by a mosquito.[3]

The epidemics of recent years have resulted in a better appreciation of the early clinical signs of the disease, and the advocates of serum therapy have clearly demonstrated that it is possible, in many cases, to make a diagnosis in the preparalytic stage. Increased experience has led to the belief that many of us have, at some time, suffered from a mild febrile attack due to the virus of poliomyelitis, which has passed without any sequelæ but has conferred a lasting immunity.

The subject of immunity in this disease is still but poorly understood. One attack does not invariably confer a complete immunity, as several well-authenticated instances of a second attack have been reported at intervals varying from six to twenty years.[4][5] In one series of investigations it was found that 70% of normal adults possessed antiviral serum compared with 60% of those known to have suffered from the disease. This fact alone would seem to have an important bearing on the efficacy of serum treatment; and further doubt is cast on the method by recent experimental work by Sabin, Olitsky and Cox, which tends to show that tissue and humoral immunity may not run parallel for poliomyelitis or other virus diseases. This problem of "tissue resistance" as opposed to "serum resistance" is a comparatively new conception, and is obviously of first-rate importance.[6][7]

There is one fact in serum therapy which I have not seen adequately explained by its opponents, and that is the rapid symptomatic response which sometimes follows its administration.

The nasopharynx has long been regarded as the portal of entry for the virus of poliomyelitis as for many other diseases. Brodie produced what appeared to be conclusive evidence that the path was via the olfactory nerve to the central nervous system, and he maintained that there was no stage of a generalized infection, and that lesions appeared in the anterior horn cells before clinical signs developed. Some doubt has recently been cast on these conclusions by Sabin and Olitsky. They believe that the virus does not pass through the olfactory bulbs without leaving characteristic histological evidence, and that "in human beings this is not necessarily as frequent a pathway as is commonly held, and that nerve roots elsewhere are equally important portals of entry, possibly indirectly by way of the blood stream, to their ganglia".[8] This latter argument would appear to have some clinical backing, and I personally am loath to discard the theory of a general infection. Frequently one has seen in a family one child with a typical attack of paralysis, a second with a mild fever, while yet a third has a gastro-intestinal disturbance. It does not seem to be stretching probability too far to suppose that all three conditions are produced by the same organism.

However, regarding the nose as the highway for the virus, an attempt has recently been made to close the road to the invader. In monkeys this was done effectually by the use of the alum-picric acid spray. The method has been used extensively during recent epidemics in the United States of America, but statistics seem to show that the case incidence in sprayed and unsprayed individuals showed little difference. To be effective the treatment must be administered by a physician, and this alone tends to lessen its sphere of usefulness. Does the substance cause any permanent damage to the nasal mucous membrane? My colleagues who specialize in ear, nose and throat work tell me that this is unlikely. But we must remember that in monkeys we are dealing with a laboratory animal that is not a natural host for the disease, so that conditions cannot be held to be truly analogous. For twenty years it has been known that the nasal washings of certain people had the ability to neutralize the virus of poliomyelitis, and this is obviously a valuable defensive mechanism. Curiously enough, there is no interrelation between anti-viral serum and anti-viral nasal washings. A higher degree of defence is afforded by an abnormal mucosa with increased secretion, and Armstrong and Harrison are of the opinion that alum exerts its protective action by decreasing the permeability of the mucous membrane of the nose rather than by any specific antiseptic property.[9]

The developments of the last few years having been briefly considered, it is evident that we are still without any sure method of prevention or treatment. The greatest obstacle has been, of course, the mysterious nature of the causal organism. E. C. Rosenow's work is therefore of great interest.[10] In 1935 he maintained that the main symptoms of poliomyelitis and encephalitis could be reproduced with great regularity by suitable injections into animals of streptococci freshly isolated from human beings and from monkeys ill with the disease, and from other sources. The lesions were usually atypical. By the use of a special culture medium (autoclaved chick mash) and successive transfers the experimentally produced viruses became thoroughly

stabilized and behaved quite like the natural viruses of those diseases. Each of four strains was passed consecutively through the streptococcus to virus stage and the virus to streptococcus stage three times. Neurotropic cataphoretic velocity appears to be essential for the production of the virus phase.

If these results can be regularly established, we are on surer ground. The streptococcus is a familiar enemy, and if we can prove that he may in one stage of his life history masquerade as an invisible virus, we are in a better position to circumvent his depredations. It is interesting to know that a green streptococcus has been found in the throats of many of the patients in Victoria, though the authorities point out that there is no evidence that this organism is in any way responsible for the symptoms. "Prontosil" and anti-streptococcal serum have been given empirically, but have proved quite useless. So Dr. Mostyn Powell's recent remark seems unfortunately true:

> Once a child contracts the disease its progress, in the acute stage, is beyond our control. We can nurse the children, see to their backs and skin, provide them with respirators and intravenous therapy if they need it, and give them the inestimable benefit of early correct splinting; but the limiting of the destructive action of the virus in the delicate and highly specialized motor cells, I feel is, at the moment, utterly beyond our powers.[xiv]

Orthopædic treatment should commence as soon as the diagnosis is established. The application of the principle of physiological rest is as important here as in the case of joint disease, and, from personal experience, I am convinced that lack of early splintage is often responsible for delay of months in muscle recovery. When first seen these little patients are frequently restless and irritable, they refuse food, and their sleep is disturbed by painful muscle spasms. It is remarkable the relief that is afforded by suitable splinting and by the avoidance of all sources of reflex stimulation, such as changes of temperature, pressure of bed-clothes and unnecessary handling. At an early stage a careful examination should be made to estimate the amount of paralysis, and the findings should be carefully recorded. The treatment of infantile paralysis demands punctilious attention to detail by all who are connected with the case in the early or the late stages. In hospital practice the primary examination should not be left in the hands of inexperienced residents; but the opportunity should be taken of bedside instruction, not only of the resident staff, but also of the masseuse who will later be responsible for the reeducation. Looking through many hospital records I have been struck by the paucity of details in physical examination, and I am sure that many of you have found it impossible, by referring to a case sheet, to gather any accurate picture of what was the initial state of the patient whom you have been called on to treat. In the absence of such accurate records, with some uniform method of grading muscle power, how is it possible to assess results of one or other form of treatment? We are guided by impressions, which are notoriously inaccurate, and the summation of clinical experience, which might be of inestimable value, is negatived by our inertia.

The doctrine of splintage and rest has recently been vigorously attacked by Sister Kenny; but the evidence at present available does not indicate that the method advocated offers any solution to our problem. There is nothing new in this idea, and all too frequently we see in our clinics cases in which it has been carried out to its logical conclusion and children who have had a mild attack of poliomyelitis have remained untreated and have developed gross deformity over a period of years. Anyone who has seen many patients in the acute stage will realize that tender muscles resent movement, and any handling results in deterioration of the general condition of the patient. There is a tendency to look on a muscle and its nerve as things separate and apart. They should be regarded as one integral unit. Developmentally, muscles are laid down as a delicate nerve network which can be discerned before any muscle fibres, as such, are visible. With this conception in mind, one can appreciate how readily loss of conductivity can arise from a stretch neuritis of the ultimate nerve-muscle units. This, to my mind, affords the rationale for treatment of paralysed muscles in a position of relaxation.

It has been argued that splintage is harmful, in that it damages unparalysed muscles and causes stiffness of joints; but our experience shows that this is not true. Children with Pott's disease, treated for years on frames, do not develop permanent stiffness of their legs, and muscles which are not paralysed retain their inherent power of recovery certainly for as long as twenty years. We have all seen patients with foot-drop or hand-drop of long standing who have recovered after immobilization in the position of relaxation.

In assessing values, it is worth while to hark back to the teachings of some of the older orthopædic surgeons, observant clinicians with a wealth of experience. Robert Jones taught that in paralysis of the lower limbs avoidance of weight-bearing for twelve months was advisable; Lovett, in 1917, said:

> No one was more sceptical than I of the treatment by prolonged recumbency. I have become a thorough convert to the practice, not only in recent cases, but in old cases where it is desired to secure the maximum of muscular development, and have become familiar with a character of results which I had not known existed.

I do not believe that these statements can be cast into the limbo of forgotten things and regarded as out-of-date theories.

"No arbitrary time limit can be set to the process of recovery in partially paralysed muscles." I would distinguish between partially paralysed and completely paralysed muscles. My experience with patients under treatment from the acute stage over a period of years has led me to believe that muscles which with adequate treatment show no evidence of voluntary power after six to twelve months are irretrievably damaged. This point is of considerable prognostic importance, particularly in the adult patient.

When watching patients over long periods of time, I have been struck by the fact that recovery of power is not a continuous process. Improvement takes place in a series of spurts, with stationary phases in between. Even when this stationary phase is prolonged for some months, it does not mean that progress has come to an end. This type of recovery is difficult to explain, particularly in those cases in which, as Dr. Jean Macnamara has remarked, more recovery takes place in the third than in the first year.[12]

Where there has been extensive muscle involvement, splintage of the limbs in the mean position should be adopted, and careful estimations of recovery should be made at frequent intervals and the apparatus changed accordingly. Regard for muscle balance is all important, and, generally speaking, we must guard against gravity as a deforming and stretching force and favour those muscles which are of the greatest functional importance. I would suggest a greater attention to back and abdominal muscles and glutei. Minor grades of involvement of the trunk muscles, if unrecognized, can lead to gross grades of scoliosis; and a child with paralysed buttock muscles should not be allowed to lie constantly on his back and so add the effects of continuous pressure as a cause of atrophy.

With regard to splints in general, I feel strongly that more ingenuity could be exercised in their design and application. Children are frequently called upon to carry much useless weight of iron, and it should not be difficult or costly to devise more satisfactory apparatus.

In connexion with this question I believe that it is very necessary that splint workshops should be established in all the larger hospitals. This important matter has received little consideration, and we still have to wait weeks for apparatus which is often ill fitting and poorly adapted for its purpose because of the inevitable lack of cooperation between surgeon and splint maker. I know from my own experience at Shropshire Orthopædic Hospital that such workshops are economically sound, and, as well as being a valuable hospital service, they can be well utilized as a trade school for cripples and for occupational therapy in certain types of case.

I am well aware that splintage may be overdone, and a badly applied instrument may be definitely harmful. Perhaps one of the commonest faults that one notices is in the application of a caliper for a weak quadriceps. The knee straps as usually applied have a band which is tightened often inches above the patella. This is a potent cause of wasting and loss of power in the muscle. A simple horseshoe-shaped knee shield will overcome this difficulty, yet will maintain quite satisfactory extension. The same care should be exercised in the application of circular bandages to hold splints in position. I feel strongly that, so far as children are concerned, their treatment should be carried out in special institutions and they should not be exposed to the hurly-burly of a general massage department. In the Shropshire Orthopædic Hospital my results improved very much when I placed the patients in a ward by themselves and had a specially selected team of masseuses looking after them. The good reeducationist is born and not made, and some girls will never develop the necessary concentration in this type of work to get the best response from their patients. So I would advocate that patients suffering from infantile paralysis should not be distributed indiscriminately among the staff of the massage department, but should be under the care of those who show a special aptitude for the work.

It is wrong to regard the parent as an antagonist when treatment is concerned. Under present conditions it is impossible to keep all patients in hospital throughout their period of reeducation, and instruction of an intelligent mother in the exercises which the child is to perform at home will often make an amazing difference to the rate of recovery. As a side issue of this question I believe that an efficient service (on the lines of the District Nursing Association) by properly trained after-care sisters, would be of inestimable value.

In bringing this somewhat discursive paper to a close I would sum up my beliefs as follows:

That, in the absence of any specific form of therapy, it behoves us to use the methods which are known to be effective in the most efficient manner. In poliomyelitis, as in many other orthopædic conditions, this entails some reorganization of effort along lines which I have specified. If this is done I believe that we can look forward with confidence to the day when the deformities resulting from the disease (now unhappily so common) will disappear, and when the child with permanent disability will be guided through school and vocational training to take his place eventually as a contented and self-supporting member of the community.

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(12) J. Macnamara: "Paralysis of the Intrinsic Muscles of the Hand in Poliomyelitis: Use of Papier Mâché Splints", *Physiotherapy Review*, Volume XIII, March-April, 1933, page 47; *Canadian Public Health Journal*, November, 1932.

THE EARLY TREATMENT OF POLIOMYELITIS.[1]

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As it is the intention of this paper to embrace under the heading of early treatment consideration of both the preventive and therapeutic treatment of poliomyelitis, it will not be amiss to present a short résumé of the modern conception of the mode of transmission and of the pathogenesis of this disease.

Transmission.

Regarding conditions of transmission, certain facts seem to have been well established by experimental studies and epidemiological observations. The disease is caused by a filtrable virus, which enters through the nose and leaves through the nose or respiratory tract. Transmission is effected through the respiratory route by person-to-person contact, and the contact required is of the same order as that which is effective in other respiratory-borne virus diseases, such as influenza, measles and chickenpox.

The individual who is exposed to the virus, according to the amount of resistance developed, may harbour the virus in the following ways:[1] (i) without symptoms—a "carrier" state; (ii) with mild illness and symptoms of indefinite character—"possible abortive" cases; (iii) with symptoms sufficiently developed to be characteristic and recognizable by those familiar with the syndrome, although the spinal fluid may be normal —"probable abortive" cases; (iv) with characteristic clinical course and positive spinal fluid findings, but without the occurrence of demonstrable paralysis—"frank abortive" or "non-paralytic" poliomyelitis; (v) with characteristic clinical course, positive spinal fluid findings and any degree of paralysis from a transient muscle weakness to a total ascending paralysis—the typical paralytic poliomyelitis.

These classifications are not mutually exclusive, and tend to merge into each other, and because of the widely varying symptomatology and the lack of a dependable objective test, it is at present impossible to set up a satisfactory criterion of diagnosis that will include all cases of this disease. The existence of the true "carrier", namely, a healthy individual harbouring the virus, has been doubted by some investigators, as there is a lack of evidence to support the theory.

Isolation and Quarantine.

If it is accepted that the conditions of transmission are essentially as outlined, then it follows logically that the spread of the disease in human populations cannot be effectively limited by the isolation of recognized patients and their immediate contacts, no matter how rigid the restrictions imposed may be. This does not imply that such procedure should be abandoned nor that it does not in some degree restrict the spread of the disease to the immediate environment of a known case. It suffers from the same inadequacies as do isolation and quarantine in the other respiratory-borne virus diseases; that is, the case is infectious for some days before the diagnosis is made, and unrecognized patients and carriers can spread the disease just as effectively as can patients who are known to have the disease. The present Victorian epidemic, it would appear, has rather borne out this point of epidemiology. Scholes[2] reported that the epidemic was of an unusually infectious nature, and that in the early stages its spread could be followed in a district and from one district to the other, and yet attempts at curbing this spread by the normal means of isolation and quarantine were not effective.

It would seem that exposure to the virus of poliomyelitis is probably inevitable for everyone sooner or later, just as is exposure to the virus of such diseases as measles, mumps and chickenpox. Maxcy[1] suggests that preventive effort should be directed primarily not toward reduction in spread, but to decrease in the incidence of clinical attacks with their possible risks. Fortunately, in poliomyelitis the natural resistance of the vast majority of human beings to infection is sufficiently great to achieve immunity without penalty. In only one or two of each thousand of population exposed does the defence mechanism break down to the extent that paralysis results, and in only one out of each five to ten thousand or more of population is it fatal.

Prophylaxis.

Efforts have been made to improve this defence mechanism along three lines: (i) temporary passive immunity conferred by convalescent serum, (ii) active immunization by vaccination with killed or attenuated virus, and (iii) improvement in the defence mechanism at the point of entrance in the nose by means of chemical agents.

To understand satisfactorily the results of experiments along the three lines mentioned, it might be of help to discuss briefly the conception of the pathogenesis of poliomyelitis as subscribed to by the majority of workers at present. The virus is considered to be neurotropic; that is, it grows in nerve cells and propagates along their axones. The site of implantation and of initial multiplication is in the nasal mucosa, very largely, if not entirely, in the olfactory epithelium high in the nasal cavity near the cribriform plate. This the virus must reach in order to establish an effective contact. From the olfactory epithelium it propagates along the olfactory tracts to the hypothalamus. It does not appear to reach the cerebrum or the cerebellum in quantities sufficient to be demonstrable. It seems to find a better opportunity for survival by proceeding towards the brain stem and the spinal cord. It shows a particular affinity for the anterior horn cells, upon which it exerts its maximum toxic effect.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on December 9, 1937.

The systemic invasion suggested by lymphatic hyperplasia, once considered to be the primary phenomenon, is said to be secondary, due to peripheral distribution of the virus out from the central nervous system, probably along nerve pathways.

Although this conception of the pathogenesis of the disease rests largely upon observations made upon monkeys, it is consistent with knowledge derived from the study of other virus diseases and with the clinical, pathological and epidemiological manifestations of poliomyelitis in human beings.

Convalescent Serum.

The use of convalescent serum as a therapeutic agent was introduced at the time when it was held that the virus of poliomyelitis gained access to the central nervous system through the blood stream and spinal fluid. It was reasoned that if the serum was introduced early in the course of the disease, it would exert a protective effect. Flexner, Lewis and Amoss, between the years 1910 and 1917, were responsible for the experimental work on monkeys which gave rise to this theoretic basis. Serum was then given extensive clinical trial, first intrathecally and later intravenously, in small doses and later in larger doses. Its use was restricted to those patients in whom the condition was diagnosed in the preparalytic stage, on the assumption that disabling sequelæ would be prevented or ameliorated by its use before nerve cell destruction had taken place. Certain evidence was produced by Aycock[3] and his collaborators in 1928 and 1929 which suggested that the use of convalescent serum had lowered the incidence of paralysis and the mortality. It later appeared, however, that such apparent advantage in the patients treated with convalescent serum was due to the fact that the treated patients were a specially selected group of preparalytic cases, while the untreated were not. When the two groups were made as nearly alike as possible, Kramer, Aycock, Solomon and Thenebe[4] and Park[5] found that the statistical advantage in favour of the treated group disappeared.

Schultz and Gebhardt,[6] in the light of recent studies indicating that from the beginning of the infection the virus is intimately associated with neurones, and that once established in the nervous system it is propagated largely, if not entirely, along axonal routes, have reinvestigated the experimental work on monkeys. As a result of a series of carefully controlled experiments, they concluded that a highly potent serum was without demonstrable value in the treatment of experimental poliomyelitis.

With the apparent failure of clinical and experimental trial to establish the value of convalescent serum therapeutically, Kessel, Hoyt and Fisk[7] tried out its use as a prophylactic agent in an outbreak in Los Angeles. There was no evidence forthcoming to indicate that any protection was afforded. Schultz and Gebhardt[8] investigated the prophylactic question with monkeys, and found that the serum-treated animals generally developed as extensive paralysis as did the non-treated animals.

In view of this evidence, it appears that convalescent serum cannot be relied upon to confer even a temporary protection against infection.

Active Immunization.

As a result of certain limited observations on monkeys, Brodie and Kolmer decided to give their methods of vaccination a trial in human beings. Brodie[9] used an emulsion of monkey cord virus treated with formalin. This was given a careful field trial, and although it was shown to be relatively harmless, the results were in no way impressive. Kolmer[10] used a 4% "remote" monkey passage virus, treated with a 1% solution of sodium ricinoleate. This was also given a trial, and enough material was issued to immunize about 12,000 persons. Of these, ten who had received one or two doses developed poliomyelitis, the infection in five being fatal, and as convincing evidence was presented to show that the cases of paralysis following the use of Kolmer's vaccine were in fact due to the inoculated living virus and not to coincident natural exposure, the method was condemned as unsafe.

The Use of Intranasally Instilled Chemicals.

We have heard much of late about intranasally instilled chemicals. Armstrong and Harrison,[11] and later Sabin, Olitsky and Cox,[12] found that if monkeys were treated by intranasal irrigation with alum solution, a high degree of resistance was produced against subsequent intranasal instillation of virus emulsion. Schultz and Gebhardt[13] obtained similar results with numerous other chemical agents. Armstrong actually sectioned the olfactory tracts of five monkeys and then inoculated them intravenously, together with five intact controls. Four of the five controls succumbed to poliomyelitis, while the five animals whose olfactory tracts were sectioned remained well. In a similar manner, he gave intranasal instillations of picric acid in saline solution into each nostril of nine moneys; these nine prepared and nine non-treated control monkeys were then inoculated intravenously with poliomyelitis virus. Among the prepared animals there were two deaths, while among the nine controls there were six deaths from poliomyelitis. This experiment was interpreted as indicating that picric acid instilled into the nostrils tended to protect monkeys even against intravenous inoculation, and to confirm the conclusions regarding the importance of the nasal membranes and olfactory tract as a portal of entry. A 1% solution of zinc sulphate was later tried and gave even better results. This experimental evidence being accepted, there came the clinical trial. The results of several attempts in certain States in the United States of America did not produce very marked evidence of benefit. This was no doubt due to the difficulty of ensuring the application of the chemical to the desired area. It is a delicate manœuvre in the adult; in the child it can be well imagined that frequently it would be almost impossible. The spraying has to be

done at moderately frequent intervals and continued throughout the epidemic. Although many patients showed temporary ill effects of this spraying, present evidence seems to indicate that no permanent damage to the nasal mucosa results.

Epidemiological Considerations.

In Adelaide recently, Scholes,[2] of Melbourne, stated that he accepted the theory that when an epidemic of poliomyelitis came to an end the virus died and was no more. On this assumption, a further epidemic in the same country or district could be prevented by absolutely rigid quarantine. This, he pointed out, was impracticable. However, isolation of the patient and quarantine of contacts should assist in some measure in restricting the disease to the close proximity of the patient. The periods suggested for isolation of the patient and quarantine of the contacts are both three weeks. It would seem that such isolation of patients can be carried out more satisfactorily in hospitals set aside for this purpose, and, in addition to the advantage in isolation, institutional care provides better treatment and after-care in paralysed patients.

Present Position in Regard to the Use of Convalescent Serum.

In the United States of America serum is very rarely used; in Melbourne very little serum has been used during the present epidemic. In Denmark and Sweden, however, where the disease produces many cases practically every year, it is still being used, in spite of the lack of positive statistical evidence in favour of its value. In Sydney some physicians who have had considerable experience in this disease have a clinical impression that in certain cases the use of serum brings about improvement. Such improvement, they consider, is shown by a fall in temperature and by a general betterment of the condition of the patient, and has been noted so soon after the administration of the serum as to suggest that the improvement has been the direct result of its administration. The Infantile Paralysis Committee, taking into account this clinical impression, the fact that the use of serum is in no way harmful, and that no better proven method of treatment exists, considers that a supply of convalescent serum should be kept on hand and made available for use in selected cases.

The General Treatment of Patients.

The abortive and preparalytic case requires very little treatment. When diagnosis has been made and confirmed by lumbar puncture and examination of the cerebro-spinal fluid, the treatment is rest in bed. Fortunately this presents no difficulty for the nurse. The patient is sick, probably has a headache and pain in the back and the neck, wishes to lie prone, and does not desire to be disturbed. If the temperature is raised and the patient's condition causes any apprehension, convalescent serum may be administered. After a few days or a week, the back pain, neck stiffness and general malaise disappear, and the patient moves around in bed. There is very little reason why the patient should not now be permitted up. However, the isolation period of three weeks should still be enforced.

A word of warning might be given here. It occasionally happens that mild weakness of the back or of the abdominal muscles is first noted when the child gets up. Such weakness should be watched for, and steps should be taken to ensure that no deformity results. In brief, the task of the physician consists mainly in allaying the anxiety of the patient's parents and people. In this regard a wise suggestion has been made: that the use of the term "infantile paralysis" should be avoided, and the condition be referred to as "poliomyelitis".

In Sydney lumbar puncture has not been used as a therapeutic measure. It has been regarded merely as a means of confirming diagnosis, and in very few cases has more than one lumbar puncture been done. There has been a good deal of controversy as to the use of lumbar puncture. Some say that it possesses value in amelioration of symptoms, and others assert that it is actually harmful, predisposing to paralysis. As it is our custom here to remove only a quantity of cerebro-spinal fluid sufficient for testing, I have personally not noticed any particular change after puncture; that is to say, that the patient is neither better nor worse for the puncture.

One generally finds the pressure raised above 200 millimetres of cerebro-spinal fluid in the lateral position, and no more than 10 cubic centimetres are removed.

For the paralysed patient, the treatment comes under three headings: rest, prevention of deformity, and the avoidance of meddlesome therapeutics.

By rest is meant as close an approximation to the dictionary definition of the term as is possible. It means the use of a firm mattress and the correct provision of pillows. It is wise to leave the patient as much alone as possible. A practice exists among medical practitioners of examining the child in detail at each visit—testing the reflexes, holding up a paralysed limb and letting it drop. This is unnecessary, and once diagnosis has been made the rest treatment should be enforced and examinations reduced to the necessary minimum.

Under the heading of prevention of deformity comes the use of correct light splinting, as early as splints can be worn without pain. It is hardly necessary to state that the general principles in splinting are to place the limbs in a position of relaxation and comfort, in order to give absolute rest to the paralysed or weakened muscles, and to prevent the opponent muscles from over-acting. The splint must be applied to perform a specific task, and the assistance of orthopædic experts is necessary in the finer points of correct splinting.

When should reeducation of the affected muscles be started? The accepted practice is to wait some

four to six weeks before this is done. I am not sure that it might not be commenced earlier. As soon as all tenderness and pain in the muscles have disappeared (and this frequently happens long before four to six weeks), the physiotherapy staff of the institution should test the muscles in detail and rate their power. Muscle training is now instituted.

It is not my intention to discuss this section of the treatment; but the physiotherapy used comes under the headings of heat, massage and muscle training. The work must be carried out by highly skilled and well-trained workers who have a good working knowledge of anatomy and physiology. Reeducation of muscles must never be continued beyond the point of fatigue, and in this instance one might make mention of the warm pool, which is an essential part of modern treatment. Underwater training not only permits of more work with less fatigue, but serves a very useful function in improving the morale of the patient .

The tendency in poliomyelitis is towards recovery, and with skilled and devoted attention it is at times astonishing what degree of recovery takes place in what appear to be completely paralysed muscles. Further, with proper treatment, this recovery continues over long periods—two, three and even more years.

It is frequently difficult to avoid using meddlesome therapeutics, as the enforcement of the rest period conveys to the parents the impression that nothing is being done for the child. Drugs have no influence on the course of the disease, the only medication necessary being a sedative for the pain and tenderness suffered. Occasionally morphine has to be administered.

Respiratory Paralysis.

As you are aware, the incidence of paralysis of the muscles of respiration and swallowing has been unusually high in the Victorian epidemic. Patients so affected require to be nursed in a respirator, and a few of these machines have been installed at the Royal Alexandra Hospital for Children and the Prince Henry Hospital. In August, Scholes said that thirteen of the nineteen patients treated in respirators in Melbourne would recover. Without respirators, he stated, they certainly would have died. Patients suffering with respiratory difficulty in poliomyelitis appear to fall into three groups: (i) those with respiratory difficulty due to paralysis of the muscles of respiration, (ii) those with respiratory difficulty due to bulbar involvement, (iii) those in whom there is involvement of both mechanisms.

There has been considerable study of the results of respirator treatment in such cases in the United States of America, and, given in brief, it would seem that the results in Group I are satisfactory—that is, about one-half or more patients survive; the results in Group II are bad, practically all patients dying; and in Group III the results appear to be intermediate between those of Group I and Group II.[14] It is felt, therefore, that the use of the respirator is always justified in poliomyelitis for symptomatic relief, and that the best results are to be looked for in those cases with intercostal and diaphragmatic paralysis.

It is pleasing to be able to state that the respirators now being supplied by a Melbourne firm are a great advance on the original Drinker respirator, and are operating very satisfactorily. In this regard the authorities in Melbourne, who were responsible for the modifications and improvements of the original machine, are to be highly congratulated.

The nursing of a patient in a respirator calls for highly skilled and well-organized team work. The feeding, care of the skin and attention to the patient's general comfort require expert and attentive nursing, but with this it is astonishing how comfortable and contented the patients appear to be.

I fear that I have had little to say about the treatment of poliomyelitis, the subject of this paper. Perhaps the reason is that nothing we can do at present can arrest the progress of the disease. Our treatment, therefore, becomes almost symptomatic.

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MILD LEPTOSPIROSIS IN SOUTHERN QUEENSLAND: A CLASSIFICATION OF THE INFECTING LEPTOSPIRA, AND A REPORT OF EIGHT FURTHER CASES OF THE DISEASE.

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INTRODUCTION.

THE number of leptospiræ recognized as being pathogenic has increased considerably in recent years. Some of these leptospiræ, although morphologically identical with *Leptospira icterohæmorrhagiæ*, present such striking serological differences from each other and from *Leptospira icterohæmorrhagiæ* as to justify their distinction into separate types. Clinically, the diseases they cause may differ from the classical Weil's disease in many respects. Therefore, the term leptospirosis, to cover the leptospiral infections generally, is now coming into general use.

Although the classification of pathogenic leptospiræ is still incomplete, several countries are known to have a variety of types. Thus in Europe three types are recognized. These are: *Leptospira icterohæmorrhagiæ*, the cause of classical Weil's disease; *Leptospira grippo-typhosa*, causing swamp or mud fever; and *Leptospira canicola*, occurring naturally in dogs, but capable of infecting man. In Japan there are three types: *Leptospira icterohæmorrhagiæ*; *Leptospira autumnalis*, the cause of Hasamiyami or autumn fever; and *Leptospira hebdomadis*, causing Nanukayami or seven-day fever. In the Dutch East Indies, again, at least five types exist; these are the classical Weil's, Rachmat, Batavia, Baermann and Bindjei types.

In Australia there are at least four types of pathogenic leptospiræ. So far, they have not been described as occurring outside Queensland. When Weil's disease, or, more correctly, leptospirosis, occurred on the cane fields of north Queensland, it was assumed that the causal organism was *Leptospira icterohæmorrhagiæ*, in spite of the low mortality rate of the outbreaks. The recent work of Lumley,[1] however, has shown that at least two types of leptospiræ (Types *A* and *B*) are capable of causing infection. One of these (Type *B*) is distantly related to *Leptospira icterohæmorrhagiæ*, whilst the other (Type *A*) is not. Johnson, Brown and Derrick[2] have shown that *Leptospira icterohæmorrhagiæ* exists in Australia; but so far the only proved human infections have occurred in sewer workers.

In 1936, however, leptospiræ were isolated from the blood of a patient suffering from a mild febrile illness. This case occurred at Pomona, a township in the coastal district, situated eighty-six miles north of Brisbane by rail. Clayton, Derrick and Cilento[3] have reported this case, with comments on the strain of leptospira isolated.

The purpose of this article is to classify this leptospira, known provisionally as the Pomona type, into its world grouping. In addition, eight further cases of mild leptospirosis, caused by the Pomona type, are reported. Seven of these occurred during an outbreak at Beaudesert in 1937, whilst the eighth case occurred at Pomona in 1936, although finally diagnosed only in recent months. It is not proposed to deal fully with the clinical aspects of this type of leptospirosis, because a sufficiently large series of cases has not yet been collected.

Our hope is that this report of further cases of the disease may stimulate medical practitioners to forward blood from suspicious cases to the Queensland Health Department, in order that the condition may become more frequently recognized and its features better understood.

CLASSIFICATION OF POMONA TYPE OF LEPTOSPIRA.

In classifying the Pomona type of leptospira into its world group the agglutination test has been employed. Comparative virulence and immunity tests on guinea-pigs have not been employed, partly because these are not sufficiently delicate for accurate classification, and partly because these aspects have to some extent been dealt with already by Derrick.[3]

Through the generosity of Dr. W. K. Mertens, Geneeskundig Laboratorium, Batavia, and Dr. G. F. Lumley, Commonwealth Health Laboratory, Townsville, the Queensland Health Department has now acquired a series of leptospiral cultures representative of most world types. Corresponding antisera have been supplied by Dr. Mertens for most strains, and these have been supplemented by antisera prepared in the laboratory of the Queensland Health Department. With these cultures and their corresponding antisera, agglutination tests, according to the technique laid down by Davidson, Campbell, Rae and Smith,[4] have been performed. Classification tables (Tables I and II) have been compiled from these results.

These tables reveal that the Pomona type of leptospira is not identical with any type or strain kept at this laboratory. Although some cross agglutination occurs with several other types, particularly Rachmat, this relationship is not close enough to be significant. An important representative type of leptospira not included in the collection kept in this laboratory is *Leptospira hebdomadis*, the cause of seven-day fever in Japan. Professor W. A. P. Schüffner, however, as I am informed by Dr. E. H. Derrick, states that he has failed to demonstrate any significant relationship between *Leptospira hebdomadis* and the Pomona type. Therefore, pending a full and final classification of all world types of leptospiræ, it is proposed to classify the Pomona type as a separate entity.

AN OUTBREAK OF MILD LEPTOSPIROSIS IN SOUTHERN QUEENSLAND.

In the last week of March, 1937, cases of fever began to occur in the district around Beaudesert, a town situated forty miles south of Brisbane. In eight days over twenty patients were admitted to

TABLE I.

Agglutination Tests with Pomona Antiserum.

Type of Leptospira.	Dilution of Serum.								
	1/10	1/30	1/100	1/300	1/1000	1/3000	1/10000	1/30000	Control.
Pomona	+++	+++	+++	+++	++	+	+	±	–
Palembang. Sumatra strain, Rachmat type	+++	++	++	+	±	–	–	–	–
Akiyami *A*. Japanese strain of *Leptospira autumnalis*	+++	++	+	–	–	–	–	–	–
Baermann. Sumatra strain	++	+	+	±	–	–	–	–	–
Andaman. Indian strain	+	+	±	–	–	–	–	–	–
Moscow. Russian strain of *Leptospira grippo-typhosa*	+	±	–	–	–	–	–	–	–
Ballico. Australian strain, Type *A*	±	–	–	–	–	–	–	–	–
Zanoni. Australian strain, Type *B*	–	–	–	–	–	–	–	–	–
Duggan. Australian strain, classical Weil's disease type	–	–	–	–	–	–	–	–	–
Wijnberg. Netherlands strain, classical Weil's disease type	–	–	–	–	–	–	–	–	–
Roesel. Netherlands strain, dog type	–	–	–	–	–	–	–	–	–
Van Tienen. Java strain, Batavia type	–	–	–	–	–	–	–	–	–

Beaudesert district hospital. It was soon realized that all these patients were suffering from a similar type of illness. Clinically, the outstanding features of the epidemic were prostration, severe headache, and generalized muscular pain, which was most pronounced in the muscles of the abdomen. The temperature rose suddenly to a maximum of up to 40·6° C. (105° F.), and subsided fairly quickly after a period of three to eight days. Conjunctival congestion, so common in leptospirosis, was observed in several cases. In a few cases vomiting was troublesome in the early stages of the illness. Jaundice, however, was absent. A feature of the epidemic was rapid loss of weight, one patient losing 6·8 kilograms (fifteen pounds) in eight days. With the fall in temperature most of the patients made a slow but uninterrupted recovery. A rash, similar to that of "scrub" typhus, was seen in one case; two patients suffered from pain and swelling of the ankle joint.

No similar cases have occurred at Beaudesert since then.

As the clinical diagnosis was uncertain, the officers of the laboratory of the Queensland Health Department were directed to investigate the epidemic on April 9. Unfortunately, only one patient was still in hospital, the others having been discharged to their homes in a widely scattered district. Specimens of blood and urine were obtained from this patient. In addition, through the kindness of Dr. W. A. Beet and Dr. J. V. Bogle, of Beaudesert, specimens of blood were secured from six convalescent patients. Sera obtained from these specimens of blood were tested for agglutination with various organisms: *Eberthella typhosa*, *Salmonella paratyphi*, *Salmonella schottmülleri*, *Salmonella hirschfeldii*, *Proteus OX* 19, *Proteus OXK*, and *Brucella abortus*. None of the sera agglutinated any of these organisms. The sera were then tested against various strains of leptospira, and all were found to agglutinate the Pomona leptospira at a sufficiently high titre for diagnosis.

Reports of Cases.

The clinical histories of eight cases are given below, together with the results of the leptospiral agglutination tests. The first two cases are reported in some detail, but the remainder of the reports are in summarized form. Cases I and II are quoted by courtesy of Dr. J. V. Bogle; Cases III, IV, V, VI

TABLE II.

Showing Relationship of Pomona Leptospira to Various Antisera.

Antiserum.	Titre of Antiserum.		Percentage Ratio of Pomona Strain to Homologous Strain.
	Against Homologous Strain.	Against Pomona Strain.	
Palembang	3,000	300	10·0
Akiyami *A*	30,000	300	1·0
Baermann	10,000	100	1·0
Andaman	1,000	10	1·0
Moscow	3,000	10	0·3
Ballico	3,000	0	0
Zanoni	1,000	0	0
Duggan	10,000	0	0
Wijnberg	10,000	0	0
Roesel	10,000	0	0
Van Tienen	10,000	0	0

and VII by courtesy of Dr. W. A. Beet; and Case VIII by permission of Dr. G. E. B. Clayton, Pomona.

Case I.

Clinical History.—F.W.S., a male, aged twenty-six years, a dairy farmer, stated that on March 26, 1937, he had felt "off colour" in the afternoon and did not eat. He retired to bed at his usual hour (8 p.m.), but woke at 1 a.m. next day with a high fever. The patient stated that his limbs were quite stiff at that time. Being twelve miles from Beaudesert, he sent for the ambulance to bring him to hospital. On admission he was found to have a temperature of 39·1° C. (102·4° F.). During the day he complained of severe muscular pain.

On March 28 his headache, which hitherto had been slight, increased in severity until it became almost unbearable. The patient's mental condition was one of depression and irritability. Vomiting commenced also at this time and continued until next day.

On the morning of the fifth day of the illness the patient felt much improved, but he relapsed next day, when pain and swelling appeared in the left ankle joint. On the seventh day he felt better, and symptoms ceased on the eighth day. He was discharged from hospital on April 2, feeling weak, but otherwise fairly well, except for some pain in the left ankle. His temperature record is reproduced (see Figure I).

In a note dated May 16, 1937, Dr. Bogle states:

> The pain and swelling in the left ankle joint have persisted and have only slightly improved. As he had very large and septic tonsils, I have removed these with a view to helping his joint condition.

Leptospiral Agglutination Test.—Serum withdrawn on the thirty-third day from the commencement of the illness, when tested for agglutinins against various strains of leptospiræ, gave the results shown in Table III. These results indicate that the Pomona leptospira was agglutinated in a serum dilution of 1 in 10,000. No other strain is agglutinated in such a high serum dilution.

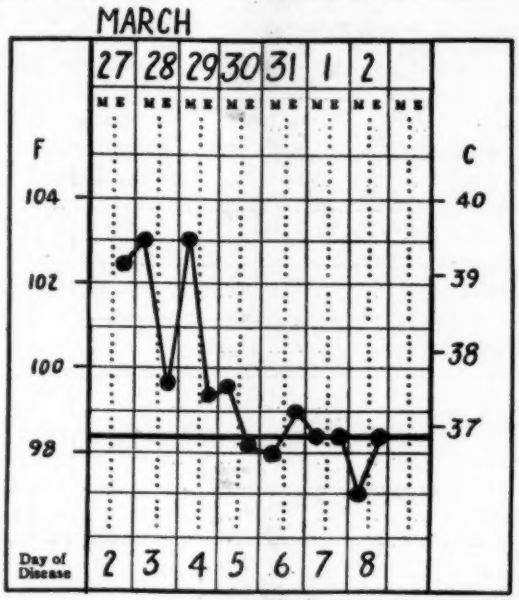

FIGURE I.
Temperature chart of patient F.W.S.

Case II.

Clinical History.—M.T.H., a male, aged thirty years, a dairy farmer by occupation, was admitted to hospital on March 28, 1937, with a history of the onset of fever and headache the previous night. He complained that the headache was very severe and that he had "cramps" in his limbs. On the second day in hospital a rash similar

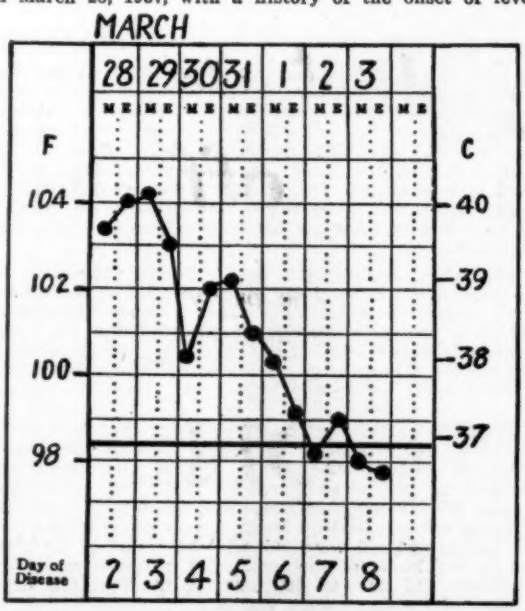

FIGURE II.
Temperature chart of patient M.T.H.

TABLE III.

Agglutination Test with Serum of F.W.S. (33rd day.)

Strain of Leptospira.	Dilution of Serum.								
	1/10	1/30	1/100	1/300	1/1000	1/3000	1/10000	1/30000	Control.
Pomona	+++	+++	+++	++	+	+	±	–	–
Palembang	+++	+++	++	+	–	–	–	–	–
Akiyami *A*	+++	++	+	±	–	–	–	–	–
Baermann	+++	++	+	±	–	–	–	–	–
Andaman	+++	+	±	–	–	–	–	–	–
Moscow	++	+	±	–	–	–	–	–	–
Ballico	+++	++	+	±	–	–	–	–	–
Zanoni	–	–	–	–	–	–	–	–	–
Duggan	–	–	–	–	–	–	–	–	–
Wijnberg	–	–	–	–	–	–	–	–	–
Rossel	–	–	–	–	–	–	–	–	–
Van Tienen	–	–	–	–	–	–	–	–	–

to that of "scrub" typhus appeared. This patient was extremely ill for a few days. He suffered from a cough without sputum and without signs of pulmonary disease. His temperature at the time of his admission to hospital was 39·7° C. (103·4° F.), rising to a maximum of 40·1° C. (104·2° F.) on the third day, thereafter falling slowly until it was normal on the eighth day of illness (see Figure II). The patient was discharged from hospital on April 3, 1937. His convalescence appears to have been uneventful.

Leptospiral Agglutination Test.—Serum withdrawn on the thirty-first day from the commencement of the illness, when tested for agglutinins against various strains of leptospiræ, gave the results shown in Table IV. The Pomona leptospira was agglutinated in a serum dilution of 1 : 30,000, whilst other strains were agglutinated either in much lower serum dilutions or not at all.

Case III.

Clinical History.—W.D., a male, aged nine years, was admitted to hospital on March 23, 1937. The patient had become ill the previous day, with vomiting, abdominal pain and severe headache, in that order of occurrence. In hospital he suffered from pains in the region of the spine and in the backs of the legs. The patient's eyes were bloodshot, and he was delirious for the first few days. In the early stages of his illness he complained most of headache and abdominal pain. There was great loss of weight. The temperature at the time of admission was 40·5° C. (105° F.). The temperature fluctuated daily, but maintained a high level until the fifth day of illness, when it commenced to fall. On the sixth day it was again raised, but finally fell to normal on the ninth day. (See Figure III.) The patient was discharged from hospital on April 1.

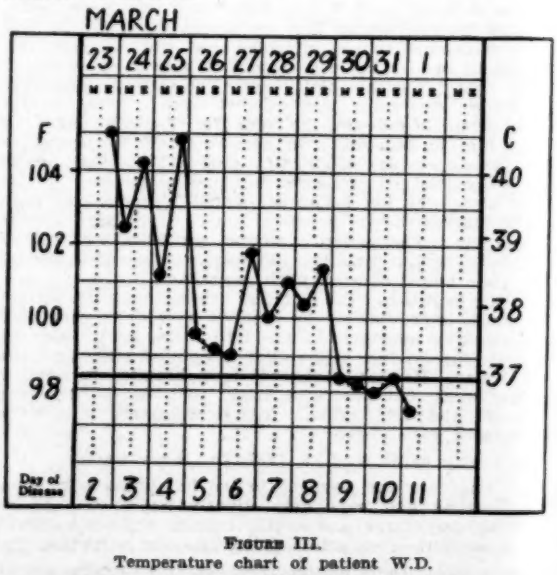

FIGURE III.
Temperature chart of patient W.D.

Leptospiral Agglutination Test.—Serum withdrawn on the thirty-sixth day from the commencement of the illness

TABLE IV.

Agglutination Tests with Serum of M.T.H. (31st day.)

Strain of Leptospira.	Dilution of Serum.								
	1/10	1/30	1/100	1/300	1/1000	1/3000	1/10000	1/30000	Control.
Pomona	+++	+++	+++	+++	+++	++	++	±	–
Palembang	+++	+++	+++	++	++	+	–	–	–
Akiyami *A*	+++	+++	++	++	+	±	–	–	–
Baermann	+++	+++	+++	+	+	±	–	–	–
Andaman	+++	++	+	±	–	–	–	–	–
Moscow	+++	++	+	±	–	–	–	–	–
Ballico	+++	++	+	±	–	–	–	–	–
Zanoni	±	–	–	–	–	–	–	–	–
Duggan	–	–	–	–	–	–	–	–	–
Wijnberg	–	–	–	–	–	–	–	–	–
Roesl	–	–	–	–	–	–	–	–	–
Van Tienen	–	–	–	–	–	–	–	–	–

agglutinated the Pomona leptospira in a dilution of 1 : 30,000.[1]

Case IV.

Clinical History.—The patient, R.D., a male, aged twelve years, was a brother of W.D. (Case III), and his history is similar. He was admitted to hospital on March 28, 1937, complaining of headache, vomiting and generalized muscular pain. The temperature was 39·4° C. (103° F.) at the time of admission, thereafter gradually falling to normal by April 2 (see Figure IV), when the patient was discharged from hospital. The duration of fever was seven days.

Leptospiral Agglutination Test.—Serum withdrawn on the thirty-first day from the commencement of the illness agglutinated the Pomona leptospira in a dilution of 1 : 30,000.

Case V.

Clinical History.—R.P., a male, aged twenty years, was admitted to hospital on March 30, 1937, complaining of headache and muscular pains. The temperature at the time of admission was 39·4° C. (103° F.), and it fell gradually until it became normal on the fifth day of the illness (see Figure V). A slight rise in temperature to 38° C. (100·4° F.) occurred on the seventh day, and the patient was discharged from hospital on April 5.

[1] In this and other cases serum agglutination test results obtained were analogous to those given in Tables III and IV (Case I and Case II). Therefore it is not proposed to quote the full agglutination results for all leptospiral strains, but only the serum titre against the Pomona strain.

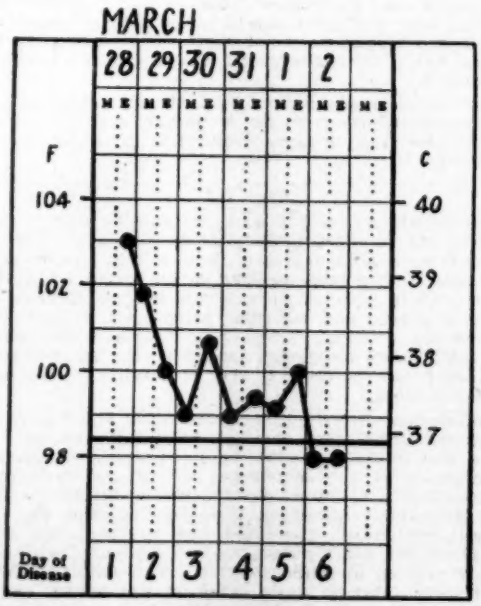

FIGURE IV.

Temperature chart of patient R.D.

Leptospiral Agglutination Test.—Serum withdrawn on the sixteenth day after the commencement of the illness agglutinated the Pomona leptospira in a dilution of 1 : 3,000. It is probable that if serum withdrawn at a later date had been tested, its titre would have been higher.

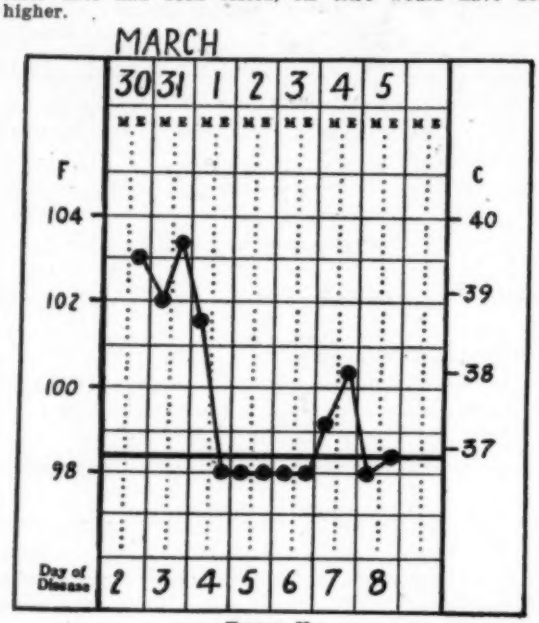

FIGURE V.
Temperature chart of patient R.P.

Case VI.

Clinical History.—D.D., a male, aged nineteen years, was admitted to hospital on March 31, 1937, complaining of a severe headache and pains all over his body. He had continual nausea for the first two days in hospital and was prostrate. The temperature reached a maximum of 39·7° C. (103·4° F.) on the first day and fell rapidly so that it was normal on the third day after admission (see Figure VI). The patient was discharged from hospital on April 5.

Leptospiral Agglutination Test.—Serum withdrawn on the thirty-third day from the commencement of the illness agglutinated the Pomona leptospira in a dilution of 1 : 10,000.

Case VII.

Clinical History.—J.P.R., aged twenty-nine years, a dairy farmer, was admitted to hospital on March 30, 1937, suffering from a mild degree of furunculosis. Prior to his admission he had been working on his farm. On April 2, whilst still in hospital, he suddenly became affected with severe headache and muscular pains. He was prostrate and mildly delirious. The following day photophobia and conjunctival congestion appeared. He was well again on April 6, but continued to stay in hospital for treatment for furunculosis.

Pathological Investigations.—On April 9 specimens of blood and urine were obtained from this patient. The urine was centrifuged, but no leptospiræ were seen on examination of the centrifuged deposit by dark-ground illumination. A guinea-pig inoculated intraperitoneally with 4·0 cubic centimetres of urine died next day from general peritonitis. The blood was separated into serum and blood clot, and a guinea-pig was inoculated with blood clot ground up in saline solution. This animal failed to react after fourteen days, when it was killed, and an emulsion of liver and kidney tissue was injected into a second passage animal. This guinea-pig likewise failed to react. The serum, when tested for agglutinins against various strains of leptospiræ, agglutinated the Pomona leptospira in a dilution of 1 : 100.

Conclusions.—This patient apparently became infected with leptospirosis whilst under treatment in hospital for another condition. Diagnosis was made clinically by the close resemblance to the other cases.

The specimens of blood and urine were obtained on the eighth day of illness. This is a rather unfavourable time, because the leptospiræ have probably disappeared from the blood stream and have not yet begun to be excreted in the urine. Furthermore, agglutinins are at a low level until the tenth day of illness.

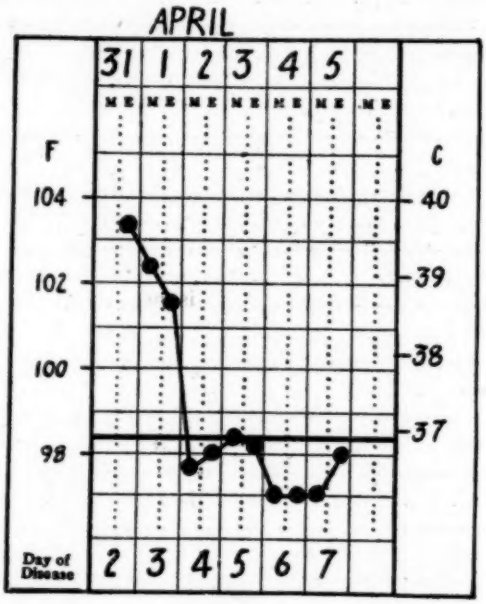

FIGURE VI.
Temperature chart of patient D.D.

Case VIII.

Clinical History.—W.F.H., an adult male, a dairy farmer by occupation, was admitted to hospital on January 26, 1936, but had been ill for three or four days prior to this. The patient complained of languor, pains and aches in the back and leg muscles, splitting headache, and pronounced photophobia. He also had persistent vomiting and epigastric pain; but these subsided after twenty-four hours in hospital. The bowel action was normal.

The temperature fell by crisis on February 1, and the patient was discharged from hospital on the following day. On February 14 the patient consulted Dr. Clayton again, because he felt weak and had no appetite. He was apyrexial, but appeared to have a pronounced secondary anæmia. Two days previously he had noticed that his vision was becoming blurred. On February 16 the patient was almost completely blind. He was given an iron tonic mixture and advised to remain in bed. By February 26 his blindness was only partial, and vision became normal a few days later. Dr. Clayton further states:

> I saw him again on March 4 and March 19, and on the latter occasion, on testing his eyes, I found he had a normal refraction and fundi. He kept out of bright sunlight and wore neutral-tint glasses for a few weeks. His recovery was absolute and he has had no eye trouble since.

Leptospiral Agglutination Test.—Serum withdrawn on August 23, 1937, that is, twenty-one months after the patient's illness, agglutinated the Pomona leptospira in a

dilution of 1 : 300. The titre of the serum is quite consistent with an active infection early in 1936. No other organism or strain of leptospira was agglutinated by this serum.

Discussion of the Cases.

Certain features of the reported cases are worthy of further consideration.

The Characteristics of the Beaudesert Epidemic.—Although only seven patients in the Beaudesert outbreak were proved to be suffering from leptospirosis, it can be assumed that the other patients (to a total of over twenty) were similarly affected. The outstanding characteristics of this outbreak were its mildness (no deaths were reported) and the absence of jaundice. Other interesting features were that all the patients were males, their ages varying from two to thirty years.

Temperature.—As no temperature records of cases of mild leptospirosis have previously been published in Australia, it was thought advisable to reproduce the charts of six patients. Study of these, however, reveals that the temperature reaction varies from case to case, and that there is no diagnostic temperature type. The maximum temperature was never under 39·4° C. (103° F.), and rose as high as 40·6° C. (105° F.), whilst the duration of pyrexia was from three to eight days.

Differential Diagnosis.—The diagnosis is a difficult matter. The sudden onset of fever, with headache, prostration and muscular pains, is characteristic of many acute diseases (particularly influenza, which hitherto has probably been the usual diagnosis). If in the first few days of illness the patient has conjunctival congestion, with or without photophobia, this may be considered suggestive of leptospirosis, although it occurs also in typhus fever. The final diagnosis of leptospirosis, however, remains with a laboratory where suitable media and leptospiral cultures are kept. In this connexion it might be advisable to state that blood withdrawn during the first week of fever generally contains leptospiræ, which may be isolated by culture or animal passage. Serum agglutinins begin to appear about the sixth day of illness, and have usually reached a diagnostic level by the tenth day.

Complications.—Three types of complication occurred in this series of cases. On the third day of the illness in one case a rash like that of "scrub" typhus appeared. Until the Weil-Felix test was performed and no reaction was found, typhus fever could not be excluded. Similar mistakes may occur when typhus fever is diagnosed on clinical grounds alone.

Two patients suffered from pain and swelling of the joints, resembling acute rheumatism. One of these patients had septic tonsils, which may explain the joint involvement; but the other patient (not reported in this series) had no such cause.

The third complication noted was temporary blindness in one patient. Eye involvement is known to occur in leptospirosis, and has been observed by Kotorii[5] and others. We have observed pronounced blurring of vision, due to clouding of the vitreous and retinal hæmorrhages, in an Italian cane-cutter following leptospirosis. It would appear reasonable to assume, therefore, that the blindness of Dr. Clayton's patient was a complication of leptospirosis.

Epidemiological Considerations.

Whilst it has not been possible to investigate this outbreak fully, certain interesting features emerge. In the first place, all of the patients lived outside the town, and no cases were reported from Beaudesert itself. Secondly, all patients became ill in the last week of March. Thirdly, the patients came from a widely scattered area, east, west and south of the town of Beaudesert, and up to a distance of sixteen miles from the town. In view of these facts, therefore, it seems necessary to postulate some infection that arose suddenly throughout the district.

There is a well-recognized association between leptospirosis and water. Patients suffering from leptospirosis have invariably been working in damp or wet places, for example sewers, or have been bathing or have fallen into water by accident. In the cane fields of north Queensland, outbreaks of leptospirosis occur only after rain and not during a dry period. It is of interest, then, to study the figures concerning the rainfall at Beaudesert. These figures have been compiled from official records at the Commonwealth Weather Bureau, Brisbane (see Tables V and VI). A study of these tables reveals the fact that the preceding year (March 1, 1936, to March 1, 1937) had been abnormally dry, whilst in March, 1937, the rainfall totalled 7·08 inches, compared with the March average of 3·68 inches. Of this total in March, 1937, no less than 5·62 inches fell over a period of four days (March 14 to 17).

TABLE V.

Rainfall at Beaudesert.

Month.	Average for Forty-four Years.	For Twelve Months from March 1, 1936, to March 1, 1937.
	Inches.	Inches.
March	3·68	3·67
April	2·53	0·26
May	1·96	1·79
June	2·22	1·91
July	1·72	1·38
August	1·21	0·16
September	1·90	1·24
October	2·37	0·66
November	2·29	1·83
December	2·49	2·49
January	5·20	1·74
February	4·91	1·96
Total	34·98	19·09

The Beaudesert district is undulating and well watered by numerous creeks. To the south and east are the Macpherson Range and the Lamington Plateau. The heavy falls of rain in the middle of March caused a local flooding of minor proportions. For a few days after this heavy fall the rural population would be working in close association with

TABLE VI.

Rainfall at Beaudesert for March, 1937.

Day of Month.	Rainfall.
	Points.
2	2
4	1
8	31
9	7
10	24
13	3
14	67
15	161
16	265
17	69
19	78
Total	708

mud and water. The population in the town of Beaudesert would not be affected to such an extent. Furthermore, all the victims were children or adult males, who were probably more exposed to infection than women.

The first case of leptospirosis occurred on March 23, and the last on April 1, 1937. This gives an incubation period of seven and fifteen days respectively from the last day on which heavy rain occurred.

In a review of thirty-seven cases of leptospirosis following a single immersion in water, Schüffner[6] gives the incubation period as from 4 to 19 days, with an average of 10·4 days. The interval elapsing between the time of heavy rain and the occurrence of cases in the Beaudesert epidemic falls within these limits, and is so striking that it cannot be attributed to coincidence. It is thus reasonable to assume that this epidemic arose as a result of heavy rain.

Regarding Case VIII, in which the illness commenced at Pomona about January 23, 1936, official records show that on January 13 the district received a heavy fall of over two inches of rain. In view of what has been said above, this rain was probably an important factor in determining the patient's illness.

Pathogenic leptospiræ can survive for a considerable time in water, especially if it is inclined to alkalinity. As an instance of this, we may quote an experiment performed by us last year. Water was taken from Kin Kin Creek in the Pomona district. This water had a hydrogen ion concentration of $10^{-7 \cdot 1}$. Five cubic centimetres of this water were boiled, and two drops of a Pomona type leptospiral culture in Schüffner's medium were added. The water was kept at room temperature (range of 6° to 24° C.). The leptospiræ multiplied considerably in this medium and did not finally disappear until thirty-three days had elapsed.

It would appear that attacks of leptospirosis in man are determined by contact with infected water. In the case of rural workers this is most likely to occur after rain.

The carrier of the Pomona type of leptospira is not yet known, but will probably prove to be a species of rat. In this connexion it is interesting to recall the statement of one patient, that in 1936 there had been quite a plague of rats in the Beaudesert district.

Much work still remains to be done before the distribution and epidemiology of this mild leptospirosis are thoroughly understood.

SUMMARY AND CONCLUSIONS.

The existence of pathogenic leptospiræ, differing serologically from *Leptospira icterohæmorrhagiæ*, is noted. The world distribution of these types is briefly discussed, and attention is drawn to the existence of four distinct types in Australia.

The Pomona type of leptospira, isolated from a human patient in southern Queensland in 1936, is tentatively classified as a new type.

Eight cases of mild leptospirosis, caused by the Pomona type of leptospira, are reported. Seven of these cases occurred during an epidemic affecting over twenty patients at Beaudesert, Queensland, in March, 1937. The eighth patient became ill at Pomona in January, 1936; but his condition was not diagnosed pathologically until August, 1937. In these eight cases the outstanding clinical features were fever, prostration, severe headache and generalized muscular pains. Delirium and conjunctival congestion were also noted in several cases. The duration of fever was from three to eight days. Jaundice was not observed and the mortality was nil. Two patients suffered from pain and swelling in the joints; one patient had a typhus-like rash, and another patient became temporarily blind some two weeks after apparent recovery.

The epidemiology of the Beaudesert outbreak is considered, and the theory is advanced that the probable precipitating factor was a heavy fall of rain about a week before the first patient became ill.

ACKNOWLEDGEMENTS.

We are indebted to Sir Raphael Cilento, Director-General of Health and Medical Services in Queensland, for permission to publish this paper, which records an investigation carried out, under instruction, for the Department of Health by its mobile unit. We are grateful to Dr. J. Coffey, Deputy-Director of Health, for his interest in this investigation; and to Dr. E. H. Derrick, Director, Laboratory of Microbiology and Pathology, for ever-ready assistance and helpful advice.

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We must also acknowledge the assistance given by Mr. A. J. Richards, Divisional Meteorologist, for the figures relating to rainfall at Beaudesert and Pomona.

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THE EFFECT OF AURICULAR FIBRILLATION ON THE COURSE OF HYPERTENSION.

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THE association of auricular fibrillation with mitral stenosis, with coronary disease and old age, and with thyreotoxicosis has been discussed frequently. The complication of other diseases by auricular fibrillation has received less attention, and it is the purpose of this paper to consider the effect of auricular fibrillation on the course of hypertension.

Varying estimates have been made of the frequency of auricular fibrillation in patients suffering from hyperpiesia. Janeway[1] found that auricular fibrillation was less frequent in the incompetent heart secondary to high blood pressure than in valvular disease, but he admitted that his patients were not followed throughout a period of years and that more lengthy observation might add to the incidence. Allbutt[2] agreed that auricular fibrillation was rare in hypertensive heart disease. Parkinson and Clark-Kennedy[3] maintained that the association of failure with high blood pressure and auricular fibrillation was uncommon and that non-rheumatic auricular fibrillation could seldom be explained as due to hyperpiesia. On the other hand, Mackenzie[4] held that in cases of degenerated arteries and long-continued high blood pressure auricular fibrillation frequently resulted in heart failure. Norris, Bazett and McMillan[5] said that in the early stages of hypertension the irregularity often consisted in premature contractions, but that later auricular fibrillation might be present. Lewis[6] wrote that "established auricular fibrillation occurs in a number of cases, but particularly late in the disease". Fahr[7] stated that auricular fibrillation was the most common form of arrhythmia associated with hypertension. In a series of 1,249 cases of hypertension reported by White[8] auricular fibrillation was present in 170, or 13·6%, being paroxysmal in 55. Flaxman[9] found that auricular fibrillation was present in 25·3% of 623 cases of essential hypertension. Fishberg[10] submits that, contrary to the opinion formerly widely held, auricular fibrillation was fairly common in essential hypertension.

Cookson[11] stated that high blood pressure occurred in cases of auricular fibrillation with insufficient frequency to entitle hypertension to be regarded as an important ætiological factor. McEachern and Baker[12] found 16·9% hypertensives among 575 consecutive patients with auricular fibrillation recognized by electrocardiographic records. Brown[13] observed that hypertension, with or without coronary disease, was an ætiological factor in 79·3% of cases of non-rheumatic or arteriosclerotic heart disease with permanent fibrillation, and 86·5% of cases with transient fibrillation. According to Brill,[14] "auricular fibrillation is most often associated with certain types of heart disease, namely, rheumatic valvulitis, particularly mitral stenosis, and hypertensive heart disease".

The arbitrary level at which hypertension is said to be present varies with different authorities. Thus the lowest levels for systolic and diastolic pressures in millimetres of mercury are 150 and 90 (White[15]), 150 and 95 (Norris, Bazett and McMillan[16]) and 160 and 100 (Cowdry[17]) respectively. Most English observers prefer these last levels, especially in the older groups of patients.

In my series of patients the blood pressure readings were obtained by placing the stethoscope over the brachial artery at the elbow and reducing the pressure in the armlet. In auricular fibrillation the blood pressure is constantly varying, but an approximation to the systolic blood pressure was obtained by taking the reading when, after the pressure in the armlet was reduced, more than an occasional sound was heard. The approximate diastolic pressure was measured at the time when many of the sharp sounds became less distinct. These estimations were easier in auricular fibrillation with infrequent or moderate ventricular rates. If readings of 160 or more millimetres of mercury, systolic pressure, and of 100 or more millimetres of mercury, diastolic pressure, were obtained, hypertension was considered to be present. The blood pressure was below these levels in three patients in this series, but they were included since the left ventricle was enlarged without other obvious cause, indicating, according to Bell and Clawson,[18] that these patients almost certainly had hypertension.

Primary renal disease was excluded by biochemical and, in fatal cases, by *post mortem* examinations. As far as possible, mitral stenosis was excluded by the absence of a history of rheumatic fever, by clinical and radiological examinations, and by autopsy.

An examination was made by myself in a large proportion of the patients.

Hypertension with Established Auricular Fibrillation.

The main observations have been made from the records of fifty cases of hypertension with established auricular fibrillation. Twenty-nine males and eighteen females were suffering from essential hypertension. The remaining three females had thyreotoxic hypertension, which Parkinson and Hoyle[19] defined as hypertension associated with

thyreotoxicosis without assuming that the hypertension was thyreogenic. Their ages ranged between forty-five and seventy-five years, the majority being in the seventh decade. The average age of onset of established fibrillation was sixty-two years.

The time of onset of auricular fibrillation was not always easy to define, but usually it was indicated by increased breathlessness and often by palpitation. In more advanced cases pronounced breathlessness and œdema often followed. There were nocturnal attacks of dyspnœa in seven cases.

Seven patients had *angina pectoris*. Anginal attacks occurred before the onset of fibrillation in five cases; in two of these the attacks of pain became more frequent and more severe soon after the establishment of fibrillation, but later were less frequent and less severe than before the onset of the abnormal rhythm. In the other three patients, the pain was less frequent or disappeared entirely after the onset of fibrillation. One of the two patients in whom *angina pectoris* followed the onset of auricular fibrillation died of congestive heart failure within five months of the first seizure; the other recently developed severe congestive failure, four months after the onset of the pain.

In sixteen cases the systolic blood pressure exceeded 200 millimetres of mercury after fibrillation became established. The blood pressure was known before and after the onset of fibrillation in eleven cases, and in only three did it fall substantially with the establishment of fibrillation.

The thyreoid gland was enlarged in four cases, in three of which there was clinical evidence of thyreotoxicosis. In the other the glandular enlargement was of a non-toxic type, both clinically and at autopsy.

Electrocardiographic Findings.

In forty-eight patients there was electrocardiographic proof of auricular fibrillation. Twenty-five of these had, in addition, left ventricular preponderance, five had bundle branch block, and one showed the features of coronary thrombosis in the *T* wave of lead I. Paroxysmal ventricular tachycardia was present in two patients after treatment with digitalis, but after withdrawal of the drug the electrocardiogram again showed only auricular fibrillation.

Radiological Findings.

Forty patients with hypertension and established fibrillation were examined with the fluoroscopic screen in the anterior and in the right and left oblique positions. Teleradiograms of some of the patients were made. There was enlargement of the left ventricle in all but one instance, and uncoiling of the aorta was usually noted. In thirteen cases there was general enlargement of the heart. With the patient in the right (first) oblique position and with barium in the œsophagus, the left auricle was considered to be slightly or moderately enlarged in sixteen patients; in fourteen of these congestion of the hila of the lung was observed.

The Course.

Thirty-three patients were living after auricular fibrillation had been established for the periods shown in Table I. Twenty months was the average period which had elapsed from the onset of fibrillation until the patients were last examined.

TABLE I.

Duration of Auricular Fibrillation.	Number of Patients Living.
Up to 1 year	15
1 to 2 years	9
2 to 3 years	6
3 to 4 years	2
Over 4 years	1

Congestive heart failure had occurred in every patient at some time during the illness. The response to treatment with rest, digitalis and diuretics was variable. Some improved sufficiently to resume light work, but in others pronounced shortness of breath on exertion and œdema of the dependent parts persisted. Of the seven patients who had paroxysmal dyspnœa, four died within two years of the first attack, one is living after ten months, another is living after two months, and one could not be traced beyond six months from the first paroxysm.

Cerebral embolism occurred in six patients, and it was doubtful in a seventh. Pulmonary embolism took place in five patients, *post mortem* proof being available in three, and radiological confirmation in another. In one patient pulmonary thrombosis was found at autopsy. Coronary thrombosis occurred in two patients at the time when fibrillation became established.

Diabetes mellitus was present in two patients.

Fourteen of the fifty patients have died. Table II shows the length of life from the time that fibrillation became established.

TABLE II.

Duration of Life After Onset of Auricular Fibrillation.	Number of Patients.
Up to 6 months	4
6 months to 1 year	3
1 to 2 years	5
2 to 3 years	0
3 to 4 years	2

The average duration of life of these patients from the onset of auricular fibrillation was sixteen months.

Seven *post mortem* examinations were made, and the following extracts are from summaries of the necropsies.

Necropsies Conducted at the London Hospital.

CASE I.—In the first case the diagnosis was heart failure, fibrosed infarct of the myocardium, cardio-vascular hypertrophy, hyperpiesia. Great hypertrophy and dilatation of the left ventricle, considerable dilatation of the right auricle and slight hypertrophy and dilatation of the other chambers of the heart were present. The heart weight was 29 ounces and the body weight was 169½ pounds. Patchy fibrosis of the left side of the interventricular septum, of the right part of the anterior wall and of the upper part of the posterior wall of the left ventricle was noted. There was no fibrosis of the right side of the interventricular septum. Great focal stenosis of the aorta and of other arteries was produced by fatty and calcareous atheroma. All organs showed chronic congestion. Very slight ischæmic fibrosis was found in the kidneys. One partly colloidal, partly parenchymatous adenoma was found in the thyreoid gland, which showed slight fibrosis.

CASE II.—In the second case the diagnosis was pulmonary embolism, thrombosis in the *inferior vena cava*, cerebral embolism, thrombus in the left auricular appendage, heart failure, cardio-vascular hypertrophy and hyperpiesia. A friable, pink, thrombotic embolus (0·3 centimetre long) was found in the right intracranial carotid artery at the origin of the middle cerebral artery; a similar thrombus was lightly adherent to the endocardium of the appendage of the left auricle of the heart. There were moderate hypertrophy and dilatation of the left ventricle and slight of the right ventricle. The heart weighed 19 ounces and the body weight was 158¾ pounds. There were slight atheroma of the wide coronary arteries and severe atheroma of the aorta and of other arteries. An *ante mortem* thrombus was lying unattached in the *inferior vena cava*; similar thrombi were present in the larger pulmonary arteries. A few recent infarcts (the largest was 2·5 centimetres in diameter) were found in the right kidney. Numerous scars of old infarcts were found in both kidneys.

CASE III.—In the third case the diagnosis was heart failure, cardio-vascular hypertrophy, hyperpiesia. Great hypertrophy and slight dilatation were seen in the left ventricle. The heart weighed 18¾ ounces and the body 137¾ pounds. Moderate atheroma was present in the coronary arteries and severe atheroma in the aorta. Thrombi in an early stage of organization were found in two atheromatous ulcers of the abdominal aorta. There was considerable ischæmic fibrosis in the kidneys. Organizing thrombus was present throughout the *inferior vena cava* and in the renal vein. A few small fragments of similar thrombus were found in the small pulmonary arteries, corresponding to poorly defined hæmorrhagic pulmonary infarcts. A stellate scar, one centimetre in diameter, marked the site of an old infarct in the spleen.

CASE IV.—In the fourth case the diagnosis was empyema, bronchopneumonia, cardio-vascular hypertrophy, hyperpiesia and *leontiasis ossium*. Considerable hypertrophy and slight dilatation of the left ventricle and slight hypertrophy and moderate dilatation of both auricles were present. The myocardium showed slight fatty degeneration and slight reticular fibrosis. The heart weighed 18¾ ounces and the body 127¾ pounds. Considerable atheroma of the aorta and of other arteries was found; the changes were most severe in the abdominal aorta. Very slight ischæmic fibrosis was present in the kidneys. Forty ounces of thick pus were present in the right pleural cavity. A solitary small area (three centimetres by two centimetres) of hæmorrhagico-purulent bronchopneumonia with abscess formation was found in the middle of the right lung. Pressure collapse was noted in the greater part of the lower lobe of the right lung and in small areas in the lower border of the right upper lobe and in the lower half of the right middle lobe. A nodular colloid goitre was present. Microscopic areas of glandular tissue were observed in the thymus.

CASE V.—In the fifth case the diagnosis was massive embolic infarction of the lungs, thrombosis in the heart and in the veins of the prostatic plexus and of the leg, heart failure, cardio-vascular hypertrophy and hyperpiesia. Moderate dilatation and hypertrophy of the left ventricle, slight dilatation and hypertrophy of the right ventricle and moderate dilatation of both auricles were present. The heart weighed 19 ounces and the body 140 pounds. An *ante mortem* thrombus (2·0 by 1·5 centimetres) was attached to the anterior wall of the left auricle; there were smaller portions of thrombus in the left auricular appendage, in the apex of the left ventricle, close to the apex and attached to the posterior wall of the left ventricle and in the right auricular appendage. Severe fatty and calcareous atheroma was found in the aorta and other arteries; it was most marked in the abdominal aorta. A recent hæmorrhagic infarct occupied the lower five-sixths of the lower lobe of the right lung. Three similar infarcts occupied the lower and posterior half of the left lower lobe; in the right middle lobe a firm brown organizing infarct was present. A thrombotic embolus was found in a secondary branch of the right pulmonary artery and in a smaller artery in the left lower lobe. Collapse of the upper anterior fringes of both lower lobes had occurred. There were bilateral brownish-yellow pleural effusions, the right being of 100 ounces and the left of four ounces. An *ante mortem* thrombus was found in the right and the left prostatic plexuses and in the lower part of the right femoral vein. An old, very scarred infarct (2·0 by 0·8 by 0·8 centimetres) was noticed in the congested spleen. There were congestion and very slight ischæmic fibrosis of the kidneys. Sixteen ounces of ascitic fluid were found in the peritoneal cavity.

Necropsies Conducted at the National Hospital for Diseases of the Heart, London.

CASE VI.—In the sixth case the diagnosis was heart failure, cardio-vascular hypertrophy, hyperpiesia, old infarcts of the heart, kidneys and spleen. All chambers of the heart were enlarged and hypertrophy of the left ventricle was noticed. No auricular clot was found. The valves were normal. The coronary arteries were pervious, but there was much patchy atheroma. The heart muscle was fibrous. An old infarct was found in the posterior wall of the left ventricle, near the apex, but there was no recent infarct. The heart weighed 21 ounces. There were terminal congestion and œdema of the lungs. Several old infarcts were present in the kidneys and in the spleen.

CASE VII.—In the seventh case the diagnosis was pulmonary thrombosis, heart failure, cardio-vascular hypertrophy and hyperpiesia. The lungs were congested and œdematous. The left pulmonary artery was occluded by thrombus—not embolus—producing a large infarct at the base of the left lung. The heart was moderately enlarged. The ventricles were enlarged, but the auricles were of normal size. No clots were found in any chamber. The valves were competent and normal, except the mitral valve, which, though competent, showed some thickening. There were no infarcts. All of the coronary arteries showed atheroma, the circumflex branch being almost completely obliterated.

Clinical Diagnoses.

The causes of the remaining seven deaths were diagnosed on clinical grounds. Congestive heart failure was present in six patients, and one had, in addition, gangrene of a leg from venous thrombosis, and another had a streptococcal infection of an œdematous lower extremity. The seventh patient had congestive heart failure some time previously, but the cause of death was a cerebral vascular accident, the exact nature of which was uncertain.

Two patients with thyreotoxic hypertension and established auricular fibrillation are attending hospital after five and a half years and after six years respectively from the onset of fibrillation. Both patients refused operation, but with the aid of digitalis they were able to continue restricted

activity. A third patient was not traced after observation for two and a half years; when last seen she was very short of breath and was œdematous.

Hypertension with Paroxysmal Auricular Fibrillation.

In hypertension with paroxysmal auricular fibrillation the series consisted of thirty-six patients. Fifteen males and six females had essential hypertension, and one male and fourteen females had thyreotoxic hypertension. All were between forty-five and seventy-five years of age. Although the onset of the paroxysms was much more commonly in the sixth decade in those with thyreotoxic hypertension, the seventh decade was the common age of onset in those with essential hypertension.

In a paroxysm of auricular fibrillation, palpitation and increased breathlessness were usually noticed. Pain was not a feature during the paroxysms, but between paroxysms *angina pectoris* was present in three patients.

The systolic blood pressure was over 200 millimetres of mercury in eleven patients between the paroxysms. The blood pressure was not recorded in all of the patients during the paroxysm, but in three a higher systolic blood pressure was registered on the resumption of normal rhythm after the paroxysm. In several others the blood pressure was the same during and after the paroxysm.

Thirteen patients had toxic goitre; one had been taking thyreoid extract in large doses some months previously, and later a goitre was found. Another had been taking thyreoid extract, but it was doubtful if the thyreoid gland was enlarged.

Electrocardiographic Findings.

The electrocardiogram established the diagnosis of auricular fibrillation in seventeen cases; in eight of these left ventricular preponderance was present also. In the other cases the diagnosis was made on the history or by clinical examination during a paroxysm.

Radiological Findings.

Twenty-seven of the series were examined with the fluoroscopic screen. Enlargement of the left ventricle was found in seventeen instances. There was enlargement of the left auricle in one case, as shown on examination of the patient in the right oblique position with barium suspension in the œsophagus. One of the thyreotoxic patients had an enlarged pulmonary artery.

The Course.

Death did not occur in any case during a paroxysm of auricular fibrillation. Four patients have been observed for more than five years, and during this time the paroxysms have continued despite therapy; but in others the paroxysms ceased either spontaneously or after treatment with quinidine. In four cases paroxysms recurred during periods varying from five months to four years before fibrillation became established. Three patients with thyreotoxic hypertension were treated surgically and afterwards the paroxysms ceased.

Three patients who suffered from hypertension and auricular fibrillation continuously for some weeks were treated with quinidine. Normal rhythm was restored in one case; this has persisted for two years, administration of quinidine being continued for that time. The systolic blood pressure of this patient rose from 120 to 170 and the diastolic from 90 to 110 millimetres of mercury on the resumption of normal rhythm. A second patient recently returned to normal rhythm after quinidine therapy; the blood pressure was approximately the same with fibrillation and with normal rhythm. Auricular fibrillation persisted in a third patient despite the administration of quinidine.

The Incidence of Auricular Fibrillation in Hypertension.

The notes of 1,000 consecutive patients with essential hypertension uncomplicated by mitral stenosis admitted to the general wards of the London Hospital have been examined. Admission was not necessarily gained because of hypertension or its complications, and often raised blood pressure was found on routine examination after admission for another disease. Seventy-three (or 7·3%) of these patients had auricular fibrillation. One hundred and ninety-two (or 19·2%) had congestive heart failure with normal rhythm, and 44 (or 4·4%) had congestive failure with auricular fibrillation at the time of examination.

Among 435 consecutive patients with auricular fibrillation admitted to these wards, hypertension uncomplicated by mitral stenosis was present in 49 (or 11·2%).

The case records of 95 consecutive patients who had had paroxysms of auricular fibrillation were examined. Hypertension uncomplicated by mitral stenosis was present in 32 (or 33·7%). Of these, 18 had essential hypertension and 14 had thyreotoxic hypertension.

Discussion.

The incidence of auricular fibrillation in the course of essential hypertension is difficult to determine. Flaxman[9] found auricular fibrillation in 25·3% of his cases of hypertensive heart disease; and fibrillation occurred in 13·5% of White's[8] hypertensive patients who had symptoms or signs suggestive of cardiac abnormalities. Both of these estimates are too high for hypertension as a disease, because the percentage is expressed in terms of hypertensive heart disease and not of essential hypertension. Another reason for a high figure is that White accepted systolic and diastolic blood pressures of 150 millimetres of mercury and 90 millimetres of mercury respectively, whereas I took systolic and diastolic blood pressures of 160 millimetres of mercury and 100 millimetres of mercury respectively as a minimum for the diagnosis of hypertension. On the other hand, my estimate of

7·3% is low, as every patient was not followed until death.

The percentage of cases of auricular fibrillation in which there is associated hypertension will vary from country to country, because in places where rheumatic heart disease is common the percentage will be low, and *vice versa*. In this series, 11·2% of patients with auricular fibrillation had hypertension which was uncomplicated by mitral stenosis. This figure is low because of the prevalence of rheumatism in England.

Parkinson and Campbell[20] found hypertension in 24% of patients who had had paroxysms of auricular fibrillation. In the present series, 19% of those who had paroxysmal auricular fibrillation had essential hypertension, and a further 14% had thyreotoxic hypertension. Hypertension is therefore quite common in people who have paroxysms of auricular fibrillation.

In a series of patients suffering from essential hypertension and heart failure with normal rhythm, Parkinson and Clark-Kennedy[3] found that the average age was fifty-four years. In the present series of fifty cases the average age of those with essential hypertension and established auricular fibrillation was sixty-two years. As the abnormal rhythm was of less than two years' duration in the majority of this group, the onset of auricular fibrillation could not have prolonged life for the eight years. It appears that auricular fibrillation is more likely to occur in older people with hypertension, but there is no evidence that hypertension which is of long standing is likely to be complicated by auricular fibrillation. This is well illustrated by coarctation of the aorta, an abnormality often associated with prolonged hypertension, but seldom with auricular fibrillation.

Many observers do not record the blood pressure in auricular fibrillation, since some difficulty may be experienced in the estimation, but an approximation can usually be made. In many cases in this series the systolic blood pressure remained at a raised figure after fibrillation had become established; in some instances it was over 200 millimetres of mercury. In patients in whom the systolic blood pressure fell, the diastolic pressure sometimes remained above normal. Hence the blood pressure should always be recorded in fibrillation, since it may reveal pronounced hypertension.

Radiologically, most patients in the present series had the typical findings of hypertension, namely, enlargement of the left ventricle and uncoiling of the aorta. Auricular fibrillation influences the X ray picture. Parkinson[21] stated that the effects of auricular fibrillation are shown in the course of time by enlargement of both auricles. Babey[22] pointed out that it was difficult to find pathological proof of the current view that there was an increase in the size of the auricles in pure auricular fibrillation, while radiological evidence was scanty. In hypertension with auricular fibrillation it is often difficult to assess the amount of enlargement of the left auricle in the right (first) oblique position, because, as Evans[23] has explained, the barium in the œsophagus may be displaced either by an enlarged left auricle or by the enlarged left ventricle of hypertension, the ventricle displacing the barium directly or indirectly through the auricle. Evans stated that the low position of the left ventricle, with the absence of abruptness in its upper segment, helps to separate it from the left auricle; the outline of the enlarged left ventricle in the antero-posterior view helps; but this does not exclude enlargement of the left auricle in addition to that of the left ventricle. Babey[22] said that enlargement of the left auricle associated with auricular fibrillation can be distinguished from that complicating mitral stenosis, both on clinical examination and on the absence of the square-shaped heart and enlarged pulmonary arc of mitral stenosis. In the present series the left auricle was slightly enlarged in only one case of paroxysmal fibrillation. With established fibrillation enlargement of the left auricle was demonstrated in 40% of those examined, in most of whom there was evidence of congestive failure, which was also present in other patients without enlargement of the left auricle. Enlargement of the auricles was found also at some of the *post mortem* examinations.

As regards prognosis, in essential hypertension with paroxysmal auricular fibrillation the outlook is good. Death rarely takes place in a paroxysm. The paroxysms may recur for years, but should the frequency increase fibrillation may become established. On the other hand, the paroxysms may become less frequent or cease, and this is likely with quinidine therapy. In thyreotoxic hypertension with paroxysmal fibrillation the paroxysms usually stop after subtotal thyreoidectomy.

The prognosis for those with established fibrillation is more serious. Fishberg[24] states that the onset of auricular fibrillation in hypertension shows that the myocardium is severely damaged, though sometimes such patients get along quite well for years, especially with adequate therapy. In the present series 80% of the deaths took place within two years of the onset of fibrillation, and 70% of those surviving were under observation for less than two years. The length of life after fibrillation became established was variable. One patient lived for eight years from the onset of the abnormal rhythm; there was no clue to enable recognition of such cases soon after fibrillation was established.

Flaxman[9] stated that only when fibrillation preceded and precipitated heart failure was the course of hypertension shortened considerably. This finding held in some cases in the present series, but in others the period of survival was longer than would be expected if such a generalization was true.

Cookson[11] found in his series that the average duration of life from the onset of fibrillation in the rheumatic group above the age of eighteen years was five and a quarter years; in a smaller group of non-rheumatic patients the average period of

survival was seven years from the date of onset of fibrillation. In the writer's series the mean duration of life after fibrillation became established was sixteen months in patients who subsequently died. Among the survivors the average period of observation was twenty months from the onset of fibrillation. Therefore, the average length of life is shorter after the onset of auricular fibrillation in the course of essential hypertension than in fibrillation complicating the other groups mentioned.

The two special risks of essential hypertension associated with auricular fibrillation were congestive heart failure and embolism. Congestive heart failure was a common mode of death. Often failure was present shortly after the onset of fibrillation; but rest and continued digitalis therapy may enable sufficient recovery for the patient to resume work. Other patients became ambulatory, but they were usually very short of breath, and in this condition they lived for months or even years.

Embolism is an ever-present risk, and it may be fatal. The brain and the lungs were the common sites in which the lodging of emboli was recognized clinically; the spleen, the kidneys and other organs were involved, with or without clinical manifestations. In this series the left auricle was the site of origin of cerebral emboli, but *post mortem* examination revealed that pulmonary emboli came more often from the systemic veins than from the right auricle.

The number of patients with thyreotoxic hypertension and established auricular fibrillation was too small from which to draw any conclusions.

Summary.

1. The frequency of auricular fibrillation in 1,000 cases of hypertension uncomplicated by mitral stenosis was 7·3%.

2. Hypertension uncomplicated by mitral stenosis was present in 49 (or 11·2%) of 435 cases of auricular fibrillation.

3. Hypertension uncomplicated by mitral stenosis was present in 32 (or 33·7%) of 95 patients who had had paroxysms of auricular fibrillation.

4. A study was made of 50 patients with hypertension and established auricular fibrillation. All were between forty-five and seventy-five years of age. Auricular fibrillation is commoner in older than in younger patients with hypertension.

5. The radiological appearances are described and the difficulties in interpretation are discussed.

6. Congestive heart failure and embolism were common sequelæ.

7. The response to treatment was variable. Eighty *per centum* of the deaths occurred within two years of the onset of fibrillation, but with treatment a patient might live for several years.

8. *Post mortem* appearances in seven patients are described.

9. Thirty-six patients with hypertension and paroxysmal auricular fibrillation were studied.

10. The prognosis in hypertension with paroxysmal fibrillation is better than in hypertension with established fibrillation, other things being equal.

11. Quinidine therapy often reduces the frequency of or abolishes the paroxysms.

Acknowledgements.

This investigation was suggested by Dr. John Parkinson, to whom I am indebted for making available his private and hospital case notes, and also for helpful advice and criticism. I owe thanks to Dr. Wall, to Dr. Leyton, to Dr. Rowlands, to Professor Ellis, to Dr. Donald Hunter and to Dr. William Evans for permitting me to include patients under their care at the London Hospital. I am grateful to Dr. W. W. Woods, of the Bernhard Baron Institute, London Hospital, and to Dr. Skene Keith, of the National Hospital for Diseases of the Heart, London, for the extracts from their *post mortem* records.

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Reports of Cases.

RECONSTRUCTION OF THE EXTERNAL EAR.

By R. A. MONEY,
Sydney.

THE reconstruction of the external ear, called for on account of congenital microtia, acquired deformity or traumatic removal, is a difficult surgical procedure. The normal auricle is built up of two layers of very thin, hairless skin with practically no subcutaneous tissue, supported and given shape by a thin, irregularly complicated cartilage. Skin of this type cannot be obtained from other parts of the body; and, in its entirety, costal cartilage does not form a suitable replica, even if it is so fashioned; it tends to shrink and fold up. This must be avoided at all costs. It is also very important that both ears should be the same size rather than exactly the same contour, and that the normal angle with the head should be maintained. Several methods of transferring tissues to the auditory region have been tried. One of the earliest and classical methods is that of Szymonoskie, in which the ear is formed from a single flap of mastoid skin folded back on itself, and a supportive framework of cartilage strips is inserted after healing has taken place. New, in a personal communication, has described a technique similar to this, and has also presented[1] a case in which use was made of a tube graft. Ferris Smith constructed the entire ear on the arm and, at a later date, transferred the formed ear by a tubed pedicle to its correct site.

Another method has recently been advocated by Gillies; and two cases in which it was applied have recently been described by Nattinger.[2] It is based on the use of a cartilage graft from a member of the patient's family, preferably a female, with the same blood grouping, and usually the patient's mother. The upper three-fourths of the auricular cartilage, to include the concha, is removed subcutaneously after the skin has been stripped off its surface, assisted by local infiltration anæsthesia, via an incision made along the free rolled-over border of the helix. This gives an exact foundation and form for the new ear, and is of normal thickness. The general principles of the technique are first to bury this cartilage graft in a subcutaneous pocket made large enough to accommodate it in the mastoid region, and then, by suitably planned stages, to proceed to separate the new ear away from the head by a larger Esser inlay graft over a Stent mould. The rolled-over appearance of the helix is gained by wrapping around its free border a narrow tube graft raised from the preauricular region. Several minor plastic manœuvres will complete the final appearance. The skin of the donor's ear may shrink to about half its previous size, but in a woman this defect can readily be hidden by an appropriate coiffure.

In the present case no such willing female was available, nor did such mutilation seem indicated. Accordingly, the method described by Pierce[3] was adopted and adhered to as closely as possible. The accompanying photographs will tell their own story; but the following case notes are added in further explanation.

Case History.

A male, aged sixteen years,[1] was thrown from a bolting horse in April, 1935. He was dragged some distance, and his right auricle was almost completely torn off. The wounds healed after the patient had spent six weeks in hospital. Figure I shows the condition when he was seen in August, 1936. The right external ear was missing, except for portion of the tragus, *incisura intertragica* and lobule.

[1] This patient was shown at the British Medical Association clinical evening held at the Royal Prince Alfred Hospital on June 4, 1937.

At the first operation, intratracheal anæsthesia being induced by the administration of ether by Dr. W. I. T. Hotten, a shaped and opened-out piece of costal cartilage was buried under the skin covering the mastoid process

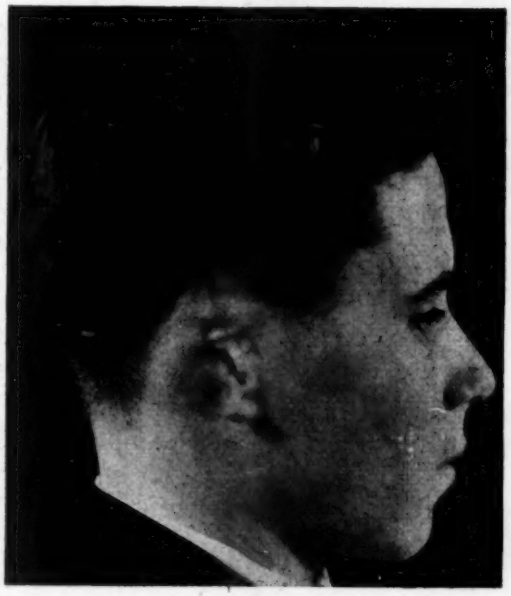

FIGURE I.

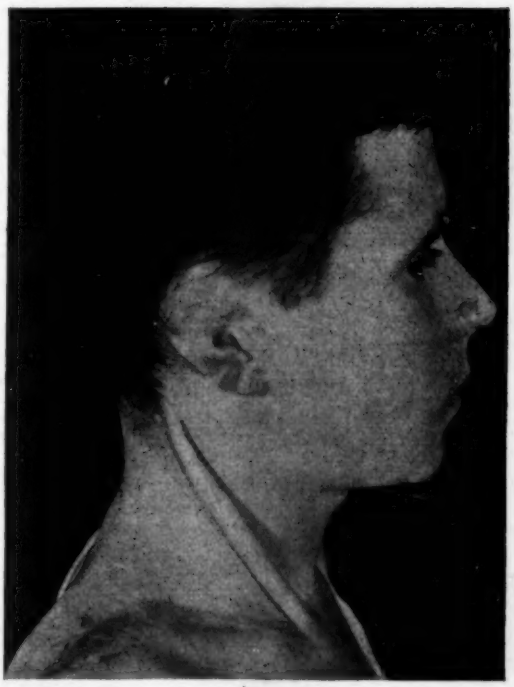

FIGURE II.

and the area above the external meatus. At the same time a long, narrow, Gillies tube-graft was raised from the right supraclavicular region. At all subsequent operations local infiltration anæsthesia was used.

Six weeks later the outer end of this tube graft was transferred to the side of the neck; this condition, together with the buried cartilage graft and the scar indicating the origin of the tube graft, can be seen in Figure II.

FIGURE III.

Four weeks later, at the third operation, the cartilage graft with the mastoid skin firmly attached to its lateral surface was raised, with the underlying temporal fascia

FIGURE IV.

attached to its medial surface, and a large Thiersch graft was placed deep to it, as an Esser inlay on a Stent mould, to cover both raw surfaces; that is, the medial surface of the new ear and the lateral surface of the mastoid region.

Two weeks later the medial end of the tube graft was transferred to the anterior and upper portion of the cartilage graft, now projecting freely from the skull. The result of this can be seen in Figure III.

At three succeeding operations the tube was partly opened out and wrapped around the outer border of the cartilage graft to form the helix and anthelix; and the original outer end was joined onto the remaining portion of the lobule. The weight of the tube caused the new ear to drop below its normal situation, and at operations performed in May and June, 1937, it was raised to its original situation. A small V-shaped portion was also excised from the left ear, which was rather unusually large, to make it conform more nearly in size to the new ear. The state of affairs at the end of June, 1937, is shown in Figures IV and V. It is intended to allow another year to elapse for the tissues to take their final shape and appearance; and then, by a series of minor

FIGURE V.

plastics, to endeavour to reduce some of the thickness of the cartilage giving the "cauliflower-ear" appearance, and also to reconstitute the hollow of the concha and the fossæ of the helix and anthelix.

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A DEATH FROM RELAPSING FEVER IN AUSTRALIA.

By J. B. CLELAND, M.D.,

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ON May 13, 1921, I carried out a *post mortem* examination at the Adelaide Hospital on a young native of India, aged about twenty years, one of the crew of a steamer belonging to the Peninsular and Oriental Steam Navigation Company. He had died one hour after admission to hospital on the day before, and pulmonary tuberculosis was suspected. The spleen was considerably enlarged, weighing 453·59 grammes (sixteen ounces), and

presented a peculiar appearance, being irregularly marbled with paler necrotic patches. The appearance of the spleen was unlike anything I had previously noticed, and a provisional diagnosis was made of splenic infarction from an unknown cause. The specimen was placed in our museum, and from time to time when I saw the specimen or came upon a reference to this patient in our records, I wondered what was the real nature of the pathological lesion.

Eleven and a half years afterwards I read an excellent account by Helen Russell on the pathology of the spleen in relapsing fever.[a] This description recalled the case in May, 1921. Sections were treated by Levaditi's method, and numerous spirochætes of relapsing fever were at once revealed. It thus took more than eleven years before the true nature of this case was revealed. It is now, five years later, perhaps wise to place the case on record before it is completely forgotten.

The notes on the spleen made at the *post mortem* examination state that it was universally enlarged, with dark congested patches, interspersed between which were irregularly marbled paler necrotic patches, often showing dark centres. On section there appeared extensive pale necrotic-looking areas, some 3·7 centimetres (one and a half inches) in diameter, others miliary in size, scattered throughout the splenic substance and occupying more than two-thirds of the tissue. The appearances suggested multiple infarcts. The glands of the hilum were dark red. The kidneys, weighing 85·05 grammes and 113·4 grammes (three ounces and four ounces) respectively, were very pale and of a yellowish tint. The liver weighed 1·219 kilograms (forty-three ounces) and was olive coloured. A number of hookworms were present in the intestines. There were no other lesions of importance.

Russell, in her description of the spleen in relapsing fever, calls attention to a zone of congestion and cell infiltration around the Malpighian bodies, which amounts almost to a collection of small abscesses. She gives a short historical survey of the pathology of the disease, and this shows that infarcts, abscesses and miliary specks are features in the pathology.

In looking through the early history of diseases in Australia, I have come upon no records which may be interpreted as of relapsing fever. It is possible that some cases of gaol fever on transports, in the early days, may have been instances of this disease. Clemow, in his "Geography of Disease", says that he has met with one reference to relapsing fever in Australia: a death was recorded in Western Australia in the year 1895.

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[a] H. Russell: "The Pathology of the Spleen in Relapsing Fever", *Transactions of the Royal Society of Tropical Medicine and Hygiene*, Volume XXVI, November, 1932, page 259.

Reviews.

PRIMITIVE INTELLIGENCE.

"PRIMITIVE INTELLIGENCE AND ENVIRONMENT", by Professor S. D. Porteus, is a very gracefully written book.[1] About half of it consists of a description of an expedition made by the author through the Kalahari Desert and adjacent regions of South Africa. In this way we are enabled to appreciate not only the difficulties of travel in that region, but also the handicaps borne by those human groups who perforce must exist there. The Kalahari is 270,000 square miles in area; it is a plateau about 3,000 feet above sea-level, with a singularly featureless surface. A great deal of it is of a sandy nature, and sand-dunes would be much more prevalent than they are, only that the sand is anchored by various grasses and thorns. The great drawback to the country is the absence of surface water from all except one part of it, Ngamiland. This prevents white settlement.

This desert is comparable in some ways to the deserts of Central and Western Australia; but Professor Porteus points out that it has an annual rainfall of from 15 to 19 inches, and undoubtedly its food supply, as far as hunting peoples are concerned, is vastly superior to that found in desert Australia. But apart from the melons and other food plants and some of the insect and animal species, some of the animals which may be eaten are themselves able and prepared to damage, kill or even eat those who would hunt them down: lions, tigers, elephants, buffalo, hippopotamus and python. Some of these, however, are to be found only in the valley of the Thamalkane River, Ngamiland, which would supply the setting for a thrilling story of adventure. The author works out the index of aridity of the Kalahari at a figure (thirteen) which puts it well above the limits for desert country, and three times as good as the Lake Eyre country of South Australia. In spite of these happier features, however, he writes: "Thorns, empty pans [claypans], sand belts, dry plains or endless swamps, heat and loneliness, thirst and starvation, and danger from wild beasts are what the Kalahari offers to those who make it their home." One wonders if, after all, life in the Kalahari would be much more delectable than in Central Australia.

Professor Porteus, however, did not make this fascinating trip into South Africa to while away a few months and to write an account of it which would arouse the wanderlust in his readers. He was concerned with the study of racial differences, and in particular with the problem of environment and intelligence. He holds the view that there are race differences in mentality, and that these are not the result of the environment working through the people's culture and social organization. He had previously (in 1929) studied the mentality of the Australian aborigines at selected sites in Central and north-western Australia, and his plan was now (in 1934) to study a people of a similar low level of hunting culture, living under similar desert conditions. He, therefore, chose the indigenous Bushmen, a pygmy people, of the Kalahari, studied their environment so as to be able to compare it with that of desert Australia; he then applied to the Bushmen the same or similar tests as those used by him with the Australian aborigines.

Unfortunately, he was able to come in contact with only a very small group of Bushmen, numbering 25. They had had very little contact with whites, and were given the tests through interpreters. The principal test used was the Porteus maze, for though, as Professor Porteus says, it "is by itself far from being a satisfactory measure of intelligence", yet he regards its performance as an index of that complex of qualities which "seem to be valuable in making adjustments to our kind of society". This is important, because it is our kind of society which is being imposed on the native, and the author holds that "the question as to who is the most intelligent by native standards is beside the mark". The results of the test were that the average score of the Bushmen was only 7·56 years as compared with the 10·52 and 12 years of the north-west and Central Australians respectively. Thus, if the environmental conditions of the two groups are similar in exhibiting the same repressive conditions, there must be definite race differences as regards mentality. The author, however, regards the Kalahari as an easier environment than the Australian, and so it may be that the supposedly harder conditions of the latter have contributed to the greater adeptness in coping with such a new condition as the coming of white culture. This principle was inherent in Professor Porteus's argument in his previous book, "The Psychology of a Primitive People"; but he seems now to have abandoned it. Possibly the results of the testing of some other groups are responsible for this.

Several Bantu groups were examined; some had been under mission influence and some not; but in all cases they were superior to the Bushmen, though just inferior to the Central Australians. As both the latter groups are failing to adapt themselves to our culture, and some of

[1] "Primitive Intelligence and Environment", by S. D. Porteus, D.Sc.; 1937. New York: The Macmillan Company. Demy 8vo, pp. 334. Price: 15s. net.

the Bantu are successful—indeed, in individual cases, they rise high—one wonders whether the maze test does supply a reliable index of adaptability to cultural change. The complexity of the situation is again emphasized by the results of the same test amongst the Sakai of the Malay Peninsula, a group which has been thought to have some affinities to the Australian aborigines, though they may in some features be compared to the South African Bushmen. They use iron, practise agriculture, build houses and make bark clothing. But, in spite of this, they scored only 10·43 years, nearly two years less than the Central Australians, though about the same as those of north-west Australia. Thus we find an agricultural people less capable (according to the test) of adaptation to our culture than the desert Australians. We do not point to these puzzles in any spirit of destructive criticism, because Professor Porteus is aware of the difficulties of his research, and at present is not prepared to claim that the results of the tests reveal anything more than fundamental race differences in mentality.

The author also tested various groups for psychophysical differences and for learning capacity, and shows the limitations and possible values of these tests. "Primitive Intelligence and Environment" will be appreciated because of its candid examination of the tests used, for its summary of the work of this type previously done in Australia, for the comparison with research carried out amongst some Asiatic primitives, for the courteous attention paid to criticisms of earlier work, and as an indication of the value of psychological testing of peoples as a means of establishing race differences and cultural adaptability or its lack.

RENAL DISEASE.

BY writing "Practical Talks on Kidney Disease", Professor Edward Weiss[1] has brought to the general practitioner and student a useful and concise survey of this difficult subject. The avowed object of the book is to help the physician to an understanding of some of the vexatious problems of disease of the kidneys. Particular attention is devoted to the practical aspects of the subject, and, in order that the book may be of greater use for those who have little time for reading, no attempt has been made to give a detailed and comprehensive survey. Despite the difficulties which brevity entails, the author has succeeded in producing a book which fulfils its object admirably. He discusses renal function in health and disease, and describes in detail the tests of renal efficiency; but it is a pity that he has not shown greater care in his description of the urea concentration test of MacLean and de Wesselow. Unless this test is carried out according to the original directions, it is liable to be misleading. The 15 grammes of urea should be given in 100 cubic centimetres of water, the volume of urine in the second specimen should not exceed 120 cubic centimetres, and if the concentration of urea is 2·5% or more it is quite certain that the kidneys are acting efficiently. Volhard's dilution and concentration test and Fishberg's modification receive the attention which their importance demands, and ample space is given to the blood urea clearance test; but the limitations of the last-named test are not discussed.

The signs and symptoms of renal disease receive adequate attention, and of special interest are the sections dealing with œdema, uræmia, cerebral manifestations and retinal lesions. By means of excellent figures and trite discussion the author points out that "albuminuric retinitis" is neither "albuminuric" nor a "retinitis", but is attributable to the lesions present in the blood vessels. All of the varied retinal changes found in association with diseases of the blood vessels and the kidneys are discussed. This section could be studied with advantage by all physicians. The author has written critical sections on glomerulonephritis, nephrosis and nephrosclerosis or arteriosclerotic disease of the kidney, and has outlined the appropriate treatment for each. An indication of the period of rest in bed necessary for patients suffering from acute nephritis, and of the signs, such as microscopic hæmaturia, which will guide the practitioner during this period, would be useful additions in any future edition. A description of the treatment of mercury poisoning by means of sodium formaldehyde sulphoxylate is a valuable feature in connexion with "mercury nephrosis", and the discussion regarding pregnancy and disease of the kidneys is instructive.

There are several printer's errors and some inelegant phrases, for example, "hourly intervals of four hours" (page 35), and "rapid delivery of œdema" (page 39); but the book will be found valuable by those who desire a brief account of the present position regarding diseases of the kidneys.

[1] "Practical Talks on Kidney Disease", by E. Weiss, M.D.; 1937. London: Baillière, Tindall and Cox. Medium 8vo, pp. 188, with illustrations. Price: 9s. net.

CLINICAL LABORATORY METHODS.

THE Standing Committee on Laboratory Methods of the University of Glasgow has issued a third edition of its "Notes on Clinical Laboratory Methods".[1] The committee which prepared these "Notes" was convened by the Dean of the Faculty of Medicine. On it the medical school and the general hospitals are equally represented, so that uniformity in the teaching of clinical laboratory methods may be safeguarded. The subject-matter of the publication is limited to those methods, mainly biochemical, which the senior medical student is required to employ in the side-rooms of the wards.

The material of this little book is marked by the compactness characteristic of similar guides for students prepared by other departments of Glasgow University. It makes no claim to completeness, but is a useful example of a desirable coordination in one branch of instruction given to medical students. While we do not suggest that teachers of different institutions should adopt standardized methods of instruction, there are certain fundamental methods for laboratory tests in which uniformity of routine would save the student much time and misunderstanding. Brief statements of the normal findings accompany many of the instructions for the performance of the tests. A few blank sheets bound at the end of the book enable the student to make comments or additions of his own.

Notes on Books, Current Journals and New Appliances.

HYGIENE FOR NURSES.

THE little manual, "Hygiene for Nurses", by Dr. J. Guy and Dr. G. J. I. Linklater,[2] appears to have been adopted as a standard text-book for nurses. The third edition was favourably reviewed in this journal about two years ago, and the fourth edition is now available. Written by the examiners to the General Nursing Council of Scotland, it contains all that a candidate need know. The examination papers set in the subject of hygiene by the General Nursing Council in the past three years are reprinted in the appendix. These show that the standard of knowledge required of Scottish nurses is high; but any candidate who has made a reasonable study of this well written book may approach her examiners with confidence.

[1] "Notes on Clinical Laboratory Methods" (Standing Committee on Laboratory Methods, University of Glasgow); Third Edition; 1936. Glasgow: John Smith and Son (Glasgow) Limited. Crown 8vo, pp. 86, with illustrations.

[2] "Hygiene for Nurses", by J. Guy and G. J. I. Linklater; Fourth Edition; 1937. Edinburgh: E. and S. Livingstone. Crown 8vo, pp. 220. Price: 5s. net.

The Medical Journal of Australia

SATURDAY, MAY 7, 1938.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

SMALLPOX AND AUSTRALIA.

THE recent occurrence of a case of smallpox among the passengers of an overseas mail steamer after its arrival in Fremantle has directed the attention of the public and of medical practitioners to this disease and to the methods that may be adopted to prevent its invasion of the Commonwealth. The distance separating Australian ports from endemic centres of smallpox is in itself a great protection, and it is perhaps not to be wondered at that the occurrence of the disease in Australia has been extremely rare. The distance from endemic centres is, of course, not the only reason for our freedom from this scourge, and he would show either ignorance or lack of appreciation who did not recognize the extraordinary efficiency of the quarantine service of the Commonwealth Department of Health. In the present instance prompt action was taken, and those who feared an outbreak of the disease had no justification for their fears. Though there is little, if any, further comment to be made on the recent occurrence, attention may well be drawn to the methods adopted for the prevention of smallpox in Australia and to the value of vaccination.

Vaccination, which is compulsory in England but not in Australia, does not, when carried out in childhood, confer lifelong immunity; revaccination from time to time is necessary. Revaccination may be successful, resulting in a fresh scar, or it may provide evidence of immunity to smallpox by producing what is known as an immunity reaction. The occurrence of an immunity reaction is accepted by quarantine authorities. A vaccinated person may thus in certain circumstances contract smallpox, but this smallpox is not so virulent as it otherwise would be, and death is not so likely to occur. In a journal such as this there is no need to lay emphasis on the value of vaccination so far as the medical profession is concerned; but the laity should have it impressed upon them that successful vaccination or the production of an immunity reaction within a period of three years should remove all fear of infection, even though the individual is passing through areas where the disease is prevalent.

It might be thought that Australia could be rendered quite safe from invasion by smallpox by the insistence that every person arriving at its ports was provided with a certificate of vaccination or of the production of an immunity reaction of recent date. This was done when regular air services with the East were instituted; but this provision entailed the compliance of the authorities of the other countries concerned and the framing of regulations in those countries. Australia cannot make and insist on regulations affecting other countries without their approval and cooperation. Air travel is so rapid, countries are brought so close together, that cooperation was easily obtained. Travel by sea is not so quick and the quarantine regulations of the Commonwealth are for all practical purposes adequate. When a steamer is quarantined considerable inconvenience and not a little expense are the lot of the shipping companies. These companies have power to make regulations and to say whom they will and whom they will not carry; they could avoid much of the inconvenience and expense if they would insist on the production by every passenger and every member of the crew

of evidence that he or she has been vaccinated within a period of, say, three years. If steamer travel becomes much more speedy it may be necessary for the Commonwealth to do as it has done with air travel; but that day is probably far distant. The lesson to the public, of course, is that overseas travelling without vaccination is foolish in the extreme.

Current Comment.

THE INTRATHECAL USE OF SULPHANILAMIDE.

IT is already difficult to keep in touch with the literature dealing with the application of the drugs of the sulphanilamide group. Among other organisms that are alleged to be vulnerable to attack by these drugs are the pneumococcus, Type III, and the meningococcus. This suggests the intrathecal administration of "Prontosil" and the other preparations of this type. A contribution to our knowledge on this subject has been made by Joseph Millett, who reports a case of pneumococcal meningitis due to a Type III infection in which sulphanilamide was given intrathecally without undue reaction.[1] He quotes the experience of other workers, one at least of whom found that the introduction of the drug into the meninges caused a pronounced local and general reaction. In Millett's case the patient did not recover and autopsy showed that while the entire body had assumed a pinkish hue due to the "Prontosil", there was nothing to indicate any chemical irritation of the brain or meninges. An unexpected finding was a large soft pituitary tumour infiltrated with pus, no doubt an important contributory cause of death; for the meninges were only moderately inflamed. During the early part of the illness the blood and cerebro-spinal fluid contained pneumococci, which grew profusely on culture; but from spinal fluid taken at intervals later in the illness only a scant growth of these organisms could be obtained. The "Prontosil" certainly seemed to have some direct germicidal effect in this case, and it diffused readily through the central nervous system. An experiment was performed on a healthy volunteer to ascertain the readiness with which the drug would find its way into the cerebro-spinal fluid after intramuscular injection. "Prontosil", in a dose of 40·0 cubic centimetres, was injected intramuscularly in two divided doses at an interval of one hour. The urine was coloured red within fifteen minutes of the first injection; but at serial lumbar taps clear fluid only was withdrawn. One hour after the second injection the cerebro-spinal fluid was still quite clear, and three hours later there was still no staining of the spinal fluid, although the subject's body was by this time coloured faintly but definitely pink by the dye. No investigation was made to discover if the cerebro-spinal fluid withdrawn under these conditions contained any sulphonamide radicals or had any bactericidal properties. It is, however, interesting to note, as the author does, that the glomeruli of the kidneys excrete the dye readily, while the chorioid plexus will not; this is consistent with some of the known differences in filtration between these two different structures. Millett concludes that the soluble form of sulphanilamide is suitable for intrathecal use and that its ready diffusibility throughout the nervous system should add to its value in meningeal infections, especially as the drug does not appear to reach the meninges, at any rate to a comparable degree, when administered after the usual fashion.

[1] *The Journal of the American Medical Association*, December 25, 1937.

THE PREVENTION AND MODIFICATION OF MEASLES.

A SATISFACTORY diminution in such infections as enteric fever, diphtheria and tuberculosis has occurred in recent years, according to Charles F. McKhann.[1] He claims, on the other hand, that there has been no fall in the incidence of measles, although the death rate has steadily lessened. The drop in the death rate is due in all probability to a recognition of the fact that the disease is particularly lethal in young children. Not only is needless exposure to the infection dangerous, but patients with measles should be isolated one from another. It is still the custom, both in the home and in some hospitals, to permit infected children to remain in close association; as all are suffering from the same disease, it is assumed that they cannot infect each other. Experience shows, however, that they can and do infect each other with such organisms as streptococci and pneumococci. Individual isolation has been followed by a lessened incidence of fatal secondary infections.

The severity of any outbreak of measles seems to depend upon the amount of resistance possessed by the community at the time of the epidemic, and upon some incalculable variation in the virulence of the virus. The fall of mass resistance is frequently observed in populations which have long been free from the infection. The lessened death rate, however, seems to be due to the general improvement in children's health, and remains unaffected by cyclical variations in the virulence of epidemics. Moreover, serious complications, such as *otitis media*, noma and pneumonia, are becoming rarer and the reactivation of tuberculous lesions in the lungs is less frequent.

Hitherto attempts to isolate and cultivate the virus of measles have been unsuccessful, though

[1] *The Journal of the American Medical Association*, December 18, 1937.

experimental inoculation of human beings was extensively carried out about one hundred and eighty years ago. Blood in very small quantity taken from a patient with measles will reproduce the disease if injected into healthy children, and the disease so induced is in all respects similar to that acquired by natural contact. One investigator, Hermann, inoculated children less than five months old with the nasal secretions of sufferers from measles while these infants still possessed a passive immunity derived from their mothers. Since the disease so produced differed in no respect from that naturally acquired, and ran no lighter a course, this practice was deservedly abandoned. The passive immunization of children shortly after they have run the risk of catching the infection would now appear to be a useful measure in the prevention of the disease; but while the death rate is steadily falling and secondary complications are becoming steadily fewer, it seems needless to use the method as a routine procedure.

The blood of almost all patients who recover from measles contains antibodies, the titre of which is greatest in the newly convalescent. Nevertheless, at any time after infection these antibodies seem to be present in amounts large enough to exert a protective influence throughout life. An intramuscular injection of four to five cubic centimetres of convalescent's serum given to non-immune persons within the first few days of exposure to the disease produces a temporary immunity and the disease fails to develop. If an injection is given at a later date, the immunity suffices to impart only partial protection and the disease manifests itself in a modified form. Often the effect of this immunization is to prolong the incubation period, and therefore children immunized in this way should undergo a lengthened period of quarantine. The modification which may be thus achieved is not to be desired when health officers or physicians are dealing with epidemics of measles in hospitals and schools, since the attenuated form of the disease is as readily communicable as the more serious forms. The important requirement is adequate protection of the acutely ill or debilitated children so frequently found in institutions. Such children form a group amongst which the mortality rate is at its highest.

The immune serum and the whole blood of adults have been used as substitutes for convalescent serum. These forms of treatment need a larger dosage, and while the results support the belief that protective bodies exist in the blood and tissues of the majority of city dwellers, they seem to show that the protection is not so certain as that bestowed by the use of convalescent serum.

As the result of the acquired immunity of their mothers and the passage through the placenta of antibodies from mother to child, infants are protected from infection during the early months of life, as is the case in scarlet fever, diphtheria and acute anterior poliomyelitis. The discovery of immune bodies in the umbilical circulation has therefore afforded a scientific reason for the use of human placenta and placental blood as a source of antibodies for protection against infection with measles. The placental extracts may be prepared with comparative ease on a fairly large scale, and so provide an important source of serum in the form of human immune globulin, which may be used in the modification or prevention of the disease. Fewer failures seem to mark the use of placental extracts and of convalescent serum than that of adult blood. The time at which the extract is injected is a matter of great importance. Larger dosage appears to compensate to some degree for delay in administration, but once the characteristic rash has appeared no amount of injected antibody is likely to exert a protective or modifying effect. If serum is given to a child in sufficient amounts during the first five days of the incubation period, it is improbable that he will contract the disease; if treatment is delayed till the fifth to ninth day, the disease is likely to appear in a modified form. After the ninth day, and especially after appearance of the eruption, little or no benefit may be expected to follow the use of serum or extract. A consideration of the available figures shows that of all children who were given injections of extract without regard to the conditions under which they were exposed to infection with measles, 63·4% were completely protected and 30·4% suffered from the disease in a modified form, while in 5·3% the attempt at immunization failed to exert either a protective or modifying influence. The extract has been shown to retain full potency for at least a year. Its use occasionally provokes general and local reactions, but these are rarely severe. The appearance of any symptoms worthy of the description of accelerated serum sickness is almost unknown. Only three patients out of a total of 2,896 treated by McKhann manifested this therapeutic complication.

Close observation of fifty-four children who were reexposed to infection with measles a few weeks after treatment with injections of immune globulin appear to show that the immunity following the injections was insufficient to protect against a reexposure, if this took place more than fourteen days after the injection of the protective dose. There was seldom even a modification of the course and severity of the disease if the reexposure occurred later than three weeks after the injection.

McKhann concludes that while active immunization against measles seems unattainable, the best method of controlling the disease is undoubtedly the practice of isolating patients, not only from the uninfected, but from one another. This is the only method which can prevent cross-infection and the sequelæ which are the real peril in this disease. Why a naturally acquired immunity to measles should be so fully protective in the vast number of urban dwellers and why the immunity conferred by the artificial use of human immune bodies should be so short-lived are riddles which yet await solution.

Abstracts from Current Medical Literature.

GYNÆCOLOGY.

End Results of Pelvic Inflammation.

H. SCHWALM (*Monatsschrift für Geburtshülfe und Gynäkologie*, November, 1937) discusses the end results of infection of the pelvic viscera. He draws particular attention to the effects of appendicitis on the uterus and its appendages. He states that sterility, associated with salpingitis and retroflexion of the uterus, frequently follows a severe attack of appendicitis, especially when drainage has been employed. In addition, he comments on the frequency with which thickening of the parametrial tissues and of the utero-sacral ligaments can be detected. This parametritis usually occurs on the right side and is often accompanied by a dextroversion of the uterus. Such displacements are not usually associated with symptoms, though he describes a case in which this association was present.

Local Treatment of Gonorrhœa.

L. POPP (*Monatsschrift für Geburtshülfe und Gynäkologie*, December, 1937) discusses two new methods employed in the local treatment of gonorrhœa in the female. The first of these is the use of "Flavadin". Pessaries of 2% "Flavadin" were inserted into the urethral canal daily, or if this was not possible a solution of similar strength was injected. The cervical canal was irrigated with a similar solution. The average duration of treatment was 28 days, with a maximum of 72 and a minimum of 10 days. In the second method "Patentex-Intradux" was used. This is a colloidal solution of alum, boric acid, potassium oxychinolate and formalin. This solution was injected with a special catheter, and the results proved quite as satisfactory as those obtained with "Flavadin". The author believes that both these preparations mark an important advance in the treatment of gonorrhœa in women.

Treatment of Pruritus Vulvæ.

W. RIEFFERSCHIED (*Monatsschrift für Geburtshülfe und Gynäkologie*, October, 1937) analyses the results he has obtained in the treatment of *pruritus vulvæ* with local applications of follicular hormone. He states that the treatment of such patients has hitherto been very unsatisfactory. For example, two-thirds of the patients whom he treated with X rays were unrelieved. The hormonal preparation used was "Progynon Ointment", and the results were remarkable for the rapid relief of symptoms. Success was more readily obtained in patients who had not yet passed the menopause than in older patients. When kraurosis was present the results were not so good. The ointment was rubbed into the whole perineal region with the hand at night or, in severe cases, both night and morning. The tissues soon became softer and pruritus rapidly disappeared. As little is known of the pathology of the condition, the author prefers to avoid expressing any opinion as to the exact mode of action of the œstrogenic hormone.

Leucoplakia, Leucokeratosis and Carcinoma of the Cervix.

W. SCHILLER (*American Journal of Obstetrics and Gynecology*, January, 1938) discusses the relationship between leucoplakia and carcinoma. He points out that leucoplakia does not describe a definite and specific disease nor a well-defined pathological, anatomical or histological picture. The word leucoplakia does nothing but convey an optic impression of a bright white patch. Various changes in the mucous membranes lead to the formation of such white patches. In the author's opinion it would be just as rational to ask whether icterus could develop into carcinoma as to ask whether leucoplakia could develop into carcinoma. The most important fact is that leucoplakia is associated with a condition in which glycogen is almost absent from the cells. This may be a simple keratosis of the epithelium which is not malignant, or on the other hand it may have been malignant *ab initio*.

The Endocrines in Relation to Sterility and Abortion.

J. C. LITZENBERG (*The Journal of the American Medical Association*, December 4, 1937) draws attention to the disappointing results obtained by the use of gonadotropic substances in the treatment of sterility. Isolated reports of successes are without value, especially as most writers fail to realize that a number of cases spontaneously return to normal. The author is of the opinion that the administration of thyreoid extract is still the best endocrine treatment for sterility.

Amenorrhœa.

R. T. FRANK, M. A. GOLDBERGER, U. J. SALMON AND G. FELSHIN (*The Journal of the American Medical Association*, December 4, 1937) record their investigations of twenty-seven patients with amenorrhœa. Six of the patients were suffering from primary amenorrhœa, and twenty-one from secondary amenorrhœa. The patients were first investigated as to secondary sex characters, basal metabolic rate, blood appearances, radiological appearances of the *sella turcica*, Janney test for sugar tolerance, blood pressure, and pelvic condition. Twenty-four out of the twenty-seven showed no serious deviations from the normal. In the great majority the sole complaint was amenorrhœa. The laboratory studies included complete œstrogenic determinations, extending over at least one month. In ten cases the determination of both œstrogenic and gonadotropic factors in blood was made and the urine was examined regularly for thirty days for both œstrogenic and gonadotropic factors. As the result of these investigations the authors were forced to the conclusion that in patients with amenorrhœa there was a wide variation in the hormone state. Evidence of almost complete ovarian afunction, subthreshold function and normal activity as well as excessive activity was found. No relationship between the gonadotropic assay and the ovarian activity was demonstrable. With regard to treatment, the authors are of the opinion that no useful purpose is served by substituting the administration of œstrogens for the combination of thyreoid administration and correction of diet in the treatment of patients whose amenorrhœa is dependent upon obesity and subthyreoidism.

Cysts of the Vulva and Vagina.

J. R. GOODALL AND F. L. MCPHAIL (*The Journal of Obstetrics and Gynæcology of the British Empire*, December, 1937) criticize the accepted form of treatment of cysts of the vulva and vagina. They state that the common cysts of the female lower genital tract arise out of deranged function of normally found structures or from activity of embryonic rests. In the former group may be placed Bartholinian cysts, either simple or infected; suburethral cysts, simple or infected; and Nabothian cysts, simple or infected. In the second group are the simple vaginal cysts, congenital cysts of the broad ligaments extending down the vaginal wall, and cysts from the remnants of Gartner's ducts. The accepted form of treatment, generally by excision, is, in the authors' opinion, usually complicated and often disfiguring. They advocate, in its stead, evacuation with destruction or merely evacuation with fenestration. In their experience the vast majority of Bartholinian cysts are not gonorrhœal in origin. They advocate treatment by incision and packing, and state that the end result is an invisible scar without deformity and without any possibility of dyspareunia. In the case of suburethral cysts or abscesses, which are of fairly common occurrence, dissection may be extremely difficult. Serious damage may be done by overlooking the connexion of the cyst with the urethra. Such a connexion is almost invariably present, but may be difficult to recognize. Perineal cysts are of many varieties and may assume large proportions, in which case dissection of the sac with reconstruction of the perineum is necessary. Nabothian cysts, which are small retention cysts of the cervix, may be catarrhal or may be due to a

diffuse chronic cervical fibrosis. For the former type the authors advocate mild cauterization. In the second type cure is not to be hoped for, and the authors deprecate strongly any attempt at treatment by cauterization and evacuation.

OBSTETRICS.

"Failed Forceps."

J. ERBSLÖH (*Monatsschrift für Geburtshülfe und Gynäkologie*, June, 1937) discusses the causation of "failed forceps" cases. He remarks on the comparative rarity of these cases in Germany as compared with those occurring elsewhere, especially in Great Britain. In a series of 20,000 deliveries only 48 such cases occurred. The maternal mortality was 9% and the fœtal mortality 58%, while the morbidity rate was very high. In the majority of patients in this series the maximum diameter of the head was still above the brim and the cervix was only partially dilated. Usually uterine inertia was stated to be the reason for the application of forceps, but in no instance had the labour lasted more than twenty-four hours. Other indications were face and brow presentations, prolapse of the cord and contracted pelvis. Trauma of the cervix and vagina was noted in many instances. Contraction ring dystocia followed the indiscriminate use of pituitary extract in eight cases. After admission to hospital the majority were delivered with forceps, while some had normal deliveries. In one instance a lower segment section was performed and on one occasion craniotomy was necessary.

Glycosuria during Pregnancy.

L. NÜRNBERGER (*Monatsschrift für Geburtshülfe und Gynäkologie*, March, 1937) has investigated the treatment of glycosuria during pregnancy with various endocrine products. He found that extracts of suprarenal gland had no real effect in such cases. The administration of *corpus luteum* preparations similarly had little effect, although good results were obtained in non-pregnant women suffering from glycosuria. The excretion of sugar during pregnancy is apparently influenced by the placenta, and the amount of sugar bears some relation to the age of the placenta.

Thyreotropic Hormone during Pregnancy.

F. BONILLA AND H. KRAMANN (*Monatsschrift für Geburtshülfe und Gynäkologie*, March, 1937) have investigated the presence of thyreotropic hormone during pregnancy. From the urine of patients with *hyperemesis gravidarum* and eclampsia a substance was obtained which caused alterations in the thyreoid glands of guinea-pigs. These changes were similar to those obtained by injection of thyreotropic hormone. Extracts of urine from normal pregnant or puerperal patients also stimulated the thyreoid gland in guinea-pigs. On the whole the investigations pointed to a close connexion between the endocrine system and the toxæmias of pregnancy.

Prolan and Œstrin in the Serum and Urine of Diabetic and Non-Diabetic Women during Pregnancy.

O. W. SMITH, in cooperation with E. P. Joslin and Priscilla White (*American Journal of Obstetrics and Gynecology*, March, 1937) reports their investigations of the œstrin and prolan content of blood and urine during pregnancy. Importance of the interplay between all the hormones is being realized more and more. It was found that prolan reached its highest peak in both urine and serum at approximately two months. By the beginning of the fifth month it reached a lower and fairly constant level. This coincided with an increase of œstrin, which reached its highest level during the last trimester. An abnormal rise in serum prolan precedes the clinical manifestation of toxæmia by at least six weeks; of eleven diabetic patients, four had either stillborn or œdematous giant infants. All four of these women at five to seven months showed the prolan-œstrin imbalance typical of toxæmia. Diabetic patients with normal curves of prolan and œstrin were delivered of normal infants.

Sudden Death during Labour.

E. LEINZINGER (*Monatsschrift für Geburtshülfe und Gynäkologie*, November, 1937) discusses the possible causes of sudden death during labour associated with marked shock and collapse. After reviewing the theories of the ætiology of shock he refers to a toxic form of which little is known. He describes a case in which this "toxic" shock occurred and in which paralysis of the peripheral circulation was evidenced by excessive hæmorrhages into the gums. This form of shock is always associated with uterine inertia, and the patients have usually had several injections of pituitary extract to stimulate contractions. He believes that the stimulating effect of such oxytocic drugs causes an increase of placental toxins in the circulation, with the consequent development of severe shock, which may prove fatal, and that therefore the amount of pituitary extract given during the first two stages of labour should be minimal. Naturally, other causes of shock and collapse have been excluded in the cases of which the histories are given.

Premature Separation of the Normally Implanted Placenta.

F. C. IRVING (*American Journal of Obstetrics and Gynecology*, November, 1937) discusses the conservative treatment of hæmorrhage from the normally implanted placenta. As the risk to the mother is greater when the hæmorrhage is external, the 353 cases are divided into two groups, according to whether the bleeding is internal or external. In the former group, comprising 234 patients, 170 were delivered normally after full dilatation. There were no deaths in this series of normal deliveries. For the last twenty years the method of controlling this type of hæmorrhage approved by most American obstetricians has been Cæsarean section. The results of treatment by Cæsarean section, in the author's opinion, cannot be considered satisfactory; for the average mortality quoted by six different authors who have treated this condition by Cæsarean section is 20·5%. He points out that a patient suffering from this type of hæmorrhage is not only handicapped by acute loss of blood, but is also the victim of shock, and in 60% of cases is suffering from toxæmia. She is therefore in a most dangerous state for surgical intervention, especially by a procedure which in itself is associated with shock and loss of blood.

Cæsarean Section.

E. M. LAZARD (*The Western Journal of Surgery, Obstetrics and Gynecology*, January, 1938) publishes his analysis of 507 consecutive cases of Cæsarean section at the Cedars of Lebanon Hospital. During the years 1930 to 1937 there were 6,068 deliveries, with a maternal death rate of 0·25%. There were 507 Cæsarean sections, constituting an incidence of 8·35%, with a maternal death rate of 1·57%. The operations were performed by forty-seven surgeons. Of the indications for Cæsarean section in this series dystocia occurred 285 times. With regard to hæmorrhage, the author does not believe that Cæsarean section should be the routine treatment for either *placenta prævia* or *ablatio placentæ*. In the toxæmia group, comprising 38 patients, there were 4 deaths, although in no case was section performed for eclamptic convulsions. This constituted a death rate of 10% for this group, and comprised 50% of the death rate of this series of Cæsarean sections.

Subcutaneous Emphysema following Cæsarean Section.

H. SCHLESINGER (*Monatsschrift für Geburtshülfe und Gynäkologie*, August, 1937) has made an exhaustive survey of the literature regarding the post-operative occurrence of subcutaneous emphysema. He reports a case in which it followed section for a failed trial of labour and was confined to the region of the neck and upper thoracic area. In most of the recorded cases the onset was observed about two days *post partum*, and recovery occurred within ten days. His patient also developed a unilateral suppurative parotitis, which rapidly healed after incision. The author states that there is no specific treatment for the emphysema.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on December 9, 1937, in the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, DR. L. A. DEY, the President, in the chair.

Poliomyelitis.

DR. LAURENCE MCDONALD read a paper entitled "The Treatment of Poliomyelitis" (see page 797).

DR. J. STEIGRAD read a paper entitled "The Early Treatment of Poliomyelitis" (see page 801).

DR. L. A. DEY spoke about the boy who had been referred to by Dr. Steigrad, and whose photograph in the respirator at the Royal Alexandra Hospital for Children had been shown. Dr. Dey said that the child had extensive paralysis except of the lower limbs, and had been in the respirator for seventeen days; his condition was improving. The question was whether there would be any difficulty in getting him out of the respirator, but on the day of the meeting he had been out for periods up to twenty minutes without distress. Dr. Dey then read the following extracts from a report made by Dr. S. W. G. Radcliff, medical superintendent of the Royal Alexandra Hospital, on the work being carried out in Melbourne.

At the outset I desire to pay a warm tribute to the courtesy extended to me by the medical superintendent of the Melbourne Children's Hospital, Dr. Douglas Galbraith, of the Frankston Orthopædic Hospital, and Dr. F. V. Scholes, of the Fairfield Infectious Diseases Hospital.

I must first explain that my main objectives in regard to anterior poliomyelitis were, firstly, information as to the hospital administrator's problems associated with anterior poliomyelitis, and secondly, information in regard to respirators and their operation. Such clinical information as I obtained was therefore mainly incidental to these objectives.

The Victorian epidemic of 1935 had presented novel and interesting features, both from the point of view of epidemiology and clinical manifestations.

Treatment.

Immune Serum.—Serum is used by some without any confidence, because, firstly, the parents or patient in many instances ask for its administration, and secondly, in the absence of any other known therapeutic agent administration is justified by giving the patient the benefit of the doubt. Others, however, have no faith whatever in serum and have discontinued its use.

Splinting and Muscle Reeducation.—Early and simple light splinting is adopted until all tenderness has disappeared. Muscle reeducation is then commenced, the massage and exercises being preceded by a warm bath. The temperature used varies from 80° F. to 95° F. The information was, however, volunteered that frequently children were too sleepy after a bath at 95° F. to cooperate effectively with the masseuse in the subsequent exercises, and that a lower temperature was desirable.

Respirators.—The outstanding feature in treatment as compared with previous Victorian epidemics had been the extensive use of mechanical respirators. This development had arisen, firstly, from the extensive paralysis in many patients, and secondly, the associated respiratory catarrh.

At the Melbourne Children's Hospital on the day of my visit there were two respirators in action. I was told that there had been four, but that two had been transferred to Fairfield, where I saw sixteen in use. The respirators at Fairfield represented stages in improvements which had been considered necessary and incorporated from time to time. The appearance of a sixteen-bed ward with respirators eight a side instead of beds, with air hoses, pulsators, controls, pilot lamps and switch gear, was most impressive. But still more so were the children in the respirators, obviously comfortable, of excellent colour, some reading comics held mechanically at a suitable angle, and others peacefully asleep. The picture was most convincing of the value and efficiency of the respirators.

Improvements on Earlier Respirators.

The more important improvements upon the earliest model are:

1. Means of regulating the respiratory rate. A rate of 22 to 24 per minute is the most suitable for children, though a quicker rate is necessary when pneumonia is present.

2. The depth of respiration can be regulated to provide at the peak of the inspiratory phase four to six inches on the water manometer in the case of children, and six to eight inches in the case of adults. The most important point, however, is that the manometer should read "zero" (equivalent to atmospheric pressure in the respirator) during the expiratory phase. This is achieved by coupling the pulsator valve in the vacuum line and providing a simple yet effective regulator. Only by strict attention to this point will emphysema and venous congestion be avoided.

3. Difficulty in securing an air-tight fitting of the rubber diaphragm around the neck without constriction of the cervical veins, pain and damage to the skin has been completely overcome by the use of a "Latex" rubber diaphragm, five-eighths of an inch thick, covered on the outer side with a thin layer of sheet rubber. A hole about four inches in diameter is cut for the neck, the edges being bevelled away on the sheet rubber side. Before application of the diaphragm two pieces of "Elastoplast", two inches wide and overlapping half an inch, are applied from the front of the sterno-mastoid on one side, around the back of the neck, to the front of the sterno-mastoid on the other side. The operator, having put his hands through the opening in the diaphragm, places one hand on each side of the child's head, the palms over the ears, thumbs over the eyes, and fingers over the cheeks. An assistant then works the diaphragm down over the operator's hands, which at the same time stretch and so enlarge the opening in the diaphragm.

4. The respirators can be tilted. This feature became necessary, firstly, in pneumonic cases to effect drainage of exudate from the respiratory tract by gravity; secondly, to assist in nasal feeding by preventing fluid from entering the respiratory tract; and thirdly, by assisting in rectal feeding, which procedure is adopted during the first day in the respirator. In addition to the tilted position, suction apparatus and atropine have sometimes to be used. When it is intended to tilt the respirator, "Elastoplast" is placed over the acromial ends of the clavicle and acromial processes of the scapulæ. A U-shaped piece of one and three-quarter inch "Latex" rubber is placed on the inside of the lid of the respirator, so that when tilted the child's weight is comfortably and safely cushioned.

5. Bed-pan and hand-hole ports are now provided in the sides of the respirator, as it requires two nurses to render bed-pan service to these patients, during which air-tightness of the respirator must be preserved.

6. Other improvements provided are a "Latex" rubber mattress to avoid bed sores; an interior electric light; an adjustable head-rest; and four clamps only to open and close the respirator.

Nursing of cases in the respirators calls for team work of the highest order, and I do not think I have ever seen a more perfect example of this than at Fairfield. There were two children whose limit without the respirator was two minutes. Sponging of these children, with treatment of their skin *et cetera*, changing of linen, remaking of beds, therefore required some means of continuing respiration during the time so occupied. This has been achieved by a simple device originated at Fairfield, comprising a standard oxygen cylinder with regulafor valve and flow gauge connected to a face-piece equipped with a valve.

From a T-piece connexion in the tubing from the regulator to the face-piece a tube is taken and immersed in a test tube containing mercury to a depth of fifteen mils. This acts as a safety valve and indicator to the operator.

It is considered necessary to have three day and two night nurses for each five respirators, in addition to the sisters in charge.

To ensure against electrical failure, all motors are connected to two independent stores of supply, with rapid change-over switches and warning pilot lamps.

Prognosis.

I asked whether the results obtained from the use of the respirators justified their use. The reply was an emphatic "yes". Several children, after continuous occupation of respirators for periods up to two months, and then intermittently for reducing periods, had been discharged to after-care homes for treatment of paralysis of the arms and legs.

DR. R. B. WADE said that he thought the present epidemic of poliomyelitis was the largest ever experienced in Australia, since the cases totalled well over one thousand. The State of New South Wales seemed to be at the beginning of an epidemic, whether great or small, time alone would show. The number of cases in Sydney at the time of the meeting was very much greater than normal. Dr. Wade said that if an epidemic did occur, it would probably resemble the Victorian epidemic, and that great changes in the management of patients would be called for. The old procedure was to put these children in a ward with other children and think no more about infection; and in his own experience Dr. Wade had known only two cases to occur in a general ward as a result of this. The Victorian epidemic was quite a different matter—the infectivity was much greater, and doctors and nurses had been infected. It was obvious that a ward would need to be set aside for these children, and this would not be convenient in most hospitals, since no other patients would be allowed to be admitted into a ward with poliomyelitis patients. The number of hospitals taking these patients would have to be limited to perhaps two or three.

Dr. Wade wondered whether it was necessary to adopt special precautions for the nurses, such as masks, sterilized gowns *et cetera*. The question was not yet settled, but care was needed. The Victorian epidemic was characterized by a great number of respiratory cases, and fifteen or sixteen respirators were all heard at work at the Queen's Memorial Hospital for Infectious Diseases at Fairfield. Perhaps in earlier epidemics the prevalence of respiratory paralysis had not been appreciated, as all patients with respiratory paralysis had died early. Respirators were being manufactured in one, two or four units, and it would be a tragedy if the electrical supply broke down. Possibly respirators should be installed only in those hospitals where a double electrical supply was available.

Speaking of nursing, team work *et cetera*, Dr. Wade said that team work was very necessary, and great precision operated at Fairfield; there were three day nurses and two night nurses to every five children. In the present epidemic a great number of children had facial paralysis, bulbar paralysis or paralysis due to involvement of the cervical part of the spinal cord. For these patients good nursing was necessary. In some cases of bulbar paralysis of the pharyngeal muscles that he had seen it was necessary to keep a sucker in operation continuously. Dr. Wade concluded his remarks by pointing out that there were many other problems which required consideration.

DR. KAREN HELMS spoke on serum therapy in poliomyelitis. She said that the great disadvantage in serum therapy had always been that the method of standardizing the serum was so expensive as to be almost prohibitive. Serum had been widely used, but in most instances its exact antibody content had been unknown. If a drug was given in unknown amounts it would not be surprising if a statistical analysis failed to show its therapeutic value.

Madsen and Jensen, of the State Serum Institute, Copenhagen, had proposed that there should be an international standard antipoliomyelitis convalescent serum. The International Commission on Biological Standardization had approved of the use of the serum prepared by Madsen and Jensen as a standard serum. Jensen found in testing convalescent serum that in some instances 0·5 cubic centimetre of serum, diluted 1 in 75, neutralized 1,000 minimal completely paralysing doses of virus. The results of the use of serum in experimental poliomyelitis were not encouraging, but experimental poliomyelitis was not human poliomyelitis. Experimental poliomyelitis was almost invariably fatal, whereas in man the mortality rate was about 10%. Harmon, Shaughnessy and Gordon had completely exsanguinated two convalescent monkeys and transferred 180 cubic centimetres of blood to each of two monkeys which had been given a large intracerebral dose of virus. Neither monkey contracted poliomyelitis. Weyer, Park and Banzhaf had some encouraging results when they used a concentrated globulin fraction of serum with high antibody content. In a controlled experiment conducted by Aycock the statistical results failed to show that the patients treated with serum did any better than untreated controls. Aycock stated, however, that he felt that from the standpoint of the patient it still could not be said that serum should not be given. Park published the results in a series of over 900 patients, half of whom were given serum in the preparalytic stage and half left untreated. There was no evidence that serum had been of benefit. Fisher, who also reported some of Park's cases, stated that in his opinion serum should be retested on strictly alternate cases. Harmon, in reviewing the results in over 2,000 cases, was of the opinion that the occasional dramatic improvement that sometimes followed the administration of serum could not be wholly disregarded. Faber had suggested that serum might possibly interrupt the passage of virus from one neurone to another at the synapse.

Dr. Helms agreed with Kramer, who considered that there was ample evidence that serum was not of absolute value, but that whether it was of any benefit at all in the disease was as yet in doubt. Dr. Helms said in conclusion that in her opinion it was unnecessary to give serum to every preparalytic patient, but that it was advisable to give it in selected cases.

DR. J. HOETS said that the epidemic in Melbourne was extraordinarily interesting. He had been to Melbourne for a week-end and had got much information about the handling of the epidemic. All the acute cases were at Fairfield, which arrangement worked very well. The hospital had about 700 beds, of which 250 were reserved for patients with acute poliomyelitis; there were five different wards, including a respirator ward with twenty-one respirators all "in full blast". One child had been in a respirator for four months and, of the others, some were able to stay out for ten hours or more. When they were ready the children were fitted with splints manufactured at the Children's Hospital, and were transferred to other hospitals, where wards were placed at the disposal of the convalescents. A great number of children were later transferred to after-care establishments. Transport of these children had proved difficult until a system had been evolved of covering the floor of the hospital "bus" with "Latex" rubber and packing the children like sardines, when they travelled very comfortably. More and more accommodation was required. Splints were ready in about a week from the time of being ordered, and were of the standard type described by Dr. Jean Macnamara. Dr. Hoets said that this type of splint was being used deliberately, and he described its chief features. In the after-care institutions, Dr. Hoets said, masseuses were at work, treating the children for short periods at fairly frequent intervals. In certain cases the children were allowed to go home from the after-care institutions; two medical visitors had control of all patients who had been allowed to do this. They visited these children, and in certain districts they had set up clinics where the children could go for inspection and treatment. The whole thing was very well under control, and expansion went on as the number of cases increased.

Many medical men thought that New South Wales was on the brink of an epidemic, and Dr. Hoets thought that some organization like that of Melbourne should be instituted in Sydney. With regard to serum treatment, Dr. Hoets said that some practitioners thought it had no value, and others thought still that it had value but that the dosage in use had not been properly worked out. In a great number of those who did have serum treatment, an almost immediate effect was observed by the attendants; and it was a significant fact that friends and relatives of medical practitioners were having serum treatment when they contracted poliomyelitis.

DR. L. L. HOLLAND asked for guidance in the selection of suitable patients for serum therapy.

Dr. Steigrad, in reply to Dr. Wade and Dr. Hoets, said that it might be of interest that between July and October, 1937, six cases of poliomyelitis had occurred in Sydney; this incidence was not much higher than normal. In November twelve cases, and in December, to the time of the meeting, six cases had occurred. He had not observed the symptoms described by Dr. Scholes, and there had been few patients with involvement of the upper regions of the central nervous system. Five patients only had facial and palatal affection. The disease was not like that occurring in Melbourne. There had been no fatalities.

In reply to Dr. Helms, Dr. Steigrad said that if some standard of serum and serum dosage was arrived at, better clinical work would be done. It might be possible to get a sufficiently high concentration. Referring to the experiment in which two monkeys had been completely exsanguinated, Dr. Steigrad said that the implication was that inadequate dosage was being used in Australia, and that there were no means of knowing whether this was so or not. Speaking of the selection of patients for serum treatment, Dr. Steigrad said that serum should be given to the child who "had his medical attendant worried". That was to say, when there was fever, when the diagnosis had been confirmed by lumbar puncture and when the patient looked more ill than the clinical signs would suggest.

A MEETING of the New South Wales Branch of the British Medical Association was held on October 21, 1937, at Sydney Hospital. The meeting took the form of a number of clinical demonstrations by members of the honorary staff. Part of this report appeared in the issue of April 30, 1938.

Malignant Disease of a Vocal Cord.

DR. R. FRANCIS showed a man, aged fifty-five years, who had been in receipt of a war pension on account of severe mustard gassing, neurasthenia and deafness of the right ear. In 1932, during the course of a medical examination in connexion with his pension, an X ray examination of the chest suggested tuberculous fibrosis of the upper lobe of the left lung; but nothing was learned from clinical and guinea-pig tests. Four months prior to the meeting the patient complained of hoarseness only, and direct examination on September 10, 1937, revealed fullness of the hypopharynx, slight limitation of movement of the right arytenoid cartilage, and a small nodular growth on the posterior third of the right vocal cord. This was removed, and the pathologist's report was as follows:

> Thickened squamous epithelium, and in one area marked change in character of cells, with amplification of small round cells in avascular connective tissue beneath. There was no evidence of tubercular structure. The condition is malignant and not inflammatory.

In the preceding two weeks, Dr. Francis said, the patient had had severe pain, radiating to the ear, and the fullness of the hypopharynx had increased. The area on the cord showed recurrence of the growth. The cervical glands were bilaterally enlarged, the left being greater than the right. The patient was having deep X ray therapy, which was started on October 18, 1937; later, surgical removal of the growth would be attempted if possible.

Malignant Growth Treated by Deep X Ray Therapy.

DR. A. T. NISBET showed two patients suffering from malignant growths who had been treated by deep X ray therapy. The first was a female, aged thirty-six years, who for years had noticed a small lump behind the right knee. About January, 1937, it broke down and commenced to discharge. She did not give any history of injury to this area. On examination a tumour was found at the upper posterior part of the right tibia. In March, 1937, the tumour behind the knee was excised, the pathological report being that the growth was a sarcoma of high grade malignancy, probably osteogenic. In May, 1937, the patient had a full course of deep X ray therapy, receiving 2,000 *r* to each of four ports, medial and lateral, anterior and posterior, making a total of 8,000 *r*. The kilovoltage was 275; five milliampères were used; the focal skin distance was 60 centimetres; a filter of three millimetres of copper *plus* one millimetre of aluminium was used. A skiagram of the upper third of the right tibia at the beginning of October, 1937, revealed no sign of newgrowth. A skiagram of the chest also revealed no abnormality.

Dr. Nisbet's second patient was a female, aged thirty-eight years. In October, 1936, she was referred, complaining of a small mass in the right breast, which had been present for six weeks. On examination a mass was found four by six by two centimetres in size, in the axillary tail of the right breast. It was mobile, hard and nodular, not attached to the skin and somewhat tender on palpation. Some enlarged glands were also seen in the right axilla, but none in the right supraclavicular region. A skiagram of the chest at this time revealed no abnormality. In October and November, 1936, the sole treatment was one course of deep X ray therapy; the patient received 2,400 *r* to each of four ports, namely, the right breast medial and lateral tangential, and the anterior and posterior axillary and supraclavicular areas. The kilovoltage was 275, five milliampères were used through a Thoreaus filter, the focal skin distance being 60 centimetres. At the time of meeting the patient had only a small, hard, fibrotic nodule, the size of the tip of a small finger, in the site of the original mass, and no axillary glands were palpable.

Dr. Nisbet finally showed a pathological specimen and skiagrams of a male patient, aged fifty-three years, who reported first with a large swelling in the upper end of the right arm in October, 1933. This was considered to be an osteochondroma according to the skiagraphic appearance. He was given six courses of deep X ray therapy to the various parts of the right humerus between November, 1933, and September, 1935, the lesion being kept in check during this period. In November, 1935, amputation of the right humerus was performed, and in December, 1936, metastatic deposits appeared in the pelvis. Further deep X ray therapy was given to these areas and controlled them, but in May, 1937, a skiagram of the chest revealed multiple metastases, and the patient died in August, 1937.

Radiographic Films.

DR. J. G. EDWARDS and DR. H. M. CUTLER exhibited several radiographic films. These films illustrated three cases of silicosis, the first in an early stage, the second and third in more advanced stages. It was pointed out that the typical nodular fibrosis was produced by the inhalation of dust containing silica and that no other dusts produced this appearance except asbestos. Silicosis was never recognized radiographically under five to eight years. Length of time had no relation to the X ray appearances, and from a film it could not be stated whether a man had been working in silica dust for ten or thirty years. The ordinary increase in markings seen in all labourers should not be confused with the nodular fibrosis or possible silicosis.

In contrast with the silicotic films, a film illustrating a case of extensive malignant metastasis in the lung was shown, and also a film of a case of complete calcification of the aorta and great vessels.

Two films showing enlargement of the *sella turcica* due to pituitary tumour were exhibited. Other films illustrated salivary calculus, ossification of a subdural hæmatoma, and a fracture-dislocation of the cerebral spine with extensive displacement but no symptoms beyond a stiff neck.

DR. D. G. MAITLAND showed a series of X ray films illustrating various types of gall-stones, some rare bone conditions, such as melorheostose, involving the pelvis and femur, adenocarcinoma of the caecum, and metastatic deposits in the lungs following an interscapulo-thoracic amputation of the right arm for osteogenic sarcoma. One case illustrated was that of a child, aged three days, who had passed no meconium since birth, and had abdominal distension with persistent vomiting. A barium enema was given by means of an ear syringe, and radiological examination then revealed a rudimentary type of colonic development with a reniform shadow of gas across the upper part of the abdomen. This was at first thought to be due to gaseous distension of the stomach as a result of congenital pyloric stenosis. A barium meal was then given by bottle, and the gaseous distension was shown to be due to pronounced dilatation of the duodenum and proximal jejunum. The child died soon after the examination, and at the *post mortem* examination atresia of the proximal jejunum was discovered, a fibrous band taking the place of about fifteen inches of the proximal part of the jejunum.

Anæsthetic Apparatus.

DR. HUGH HUNTER and DR. S. V. MARSHALL demonstrated the various agents and forms of apparatus used in the hospital for producing anæsthesia. They ranged from the Clover inhaler to the Heidbrink closed-circuit carbon dioxide absorption machine. The Lidwill and the Coutts insufflation ether machines, Junker's inhaler, Shipway's ether-chloroform apparatus, and the usual range of open masks, gags, tongue forceps, drop-bottles *et cetera* were included. Two late model McKesson gas anæsthesia machines were shown, one of which, the property of the hospital, exemplified the transition between the older fractional rebreathing and the newer carbon dioxide absorption principle. It was pointed out that as a compromise between such basically opposed principles its operation had not been quite satisfactory, efficiency being imperfect in both respects. The other machine, Dr. Hunter's property, was a later and greatly improved model; while incorporating the two principles it provided for their independent operation, so that each was very efficient. The machine was thus of value for general and special purposes, so that it might be used with safety by persons of ordinary competence while at the same time providing facilities for the specialist.

Dr. Hunter and Dr. Marshall pointed out that the Heidbrink was definitely a specialist's machine, requiring care and skill in handling. Like the McKesson machine, it employed the closed-circuit principle, but it had no mechanical provision for regulating fractional rebreathing. Its valves were extraordinarily sensitive; cardiac systole would lift them. It provided the immense advantage of a reserve supply of soda-lime. It had been found invaluable in such operations as pulmonectomy and œsophagectomy, and for use in poor-risk cases generally.

Among the anæsthetic agents demonstrated was cyclopropane. Results from the use of cyclopropane were stated to have been very satisfactory. Vinyl ether ("Vinesthene") was also represented. All the ordinary inhalational agents were on view, and the non-inhalational agents were represented by "Pentothal Sodium" and "Evipan Sodium" (for intravenous use), and "heavy" "Percain" (for spinal analgesia). Numerous other accessories were displayed, among which were the sphygmomanometer and arm-diaphragm outfit for measuring the blood pressure and pulse rate in circumstances in which the patient was relatively inaccessible, and the Leech pharyngeal gasway, a device constructed on the basis of casts from a large number of pharynges, and intended for use as an alternative to tracheal intubation.

Pathological Exhibits.

DR. H. K. PORTER and DR. A. A. MOON demonstrated a collection of microscopic sections of endometriomata from varied situations, such as the uterine wall, the ovary, the Fallopian tube, the appendix, and from an abdominal scar. They also showed a collection of specimens of the female reproductive organs in a fœtus of twenty weeks and in another of twenty-six weeks, as well as in children aged four days, six years and seven years. Seven points were demonstrated by Dr. Porter and Dr. Moon: (i) A rapid growth in size of the uterus took place between the twentieth and twenty-sixth week of intrauterine life, after which no appreciable increase occurred up to the age of six years. (ii) From shortly after birth to the age of six years a slight decrease in the size of the uterus occurred. No great growth took place in the organ until a year or two before puberty. (iii) Differentiation of the upper uterine segment from the lower cervical segment was not apparent up to the twenty-sixth week of intrauterine life. (iv) In all specimens the cervical segment was a little longer than the body segment, and this condition persisted until the onset of puberty. (v) The Fallopian tubes had doubled their length between the twentieth week and term. A progressive increase in length took place up to three and a half years, followed by a diminution in size up to the age of seven years. (vi) A progressive small increase in the size of the ovaries occurred from the twentieth week to the age of three and a half years. This was followed by a diminution in size up to the age of seven years. (vii) The decrease in size of all the reproductive organs between the ages of three and six years was obvious.

Other features of the infantile uterus were demonstrated: (i) In infancy the *arbor vitæ* extended from the cervix entirely up the body of the uterus. (ii) The uterine endometrium in infancy was dense and the uterine glands made their appearance between the first and seventh years of life as superficial invaginations. (iii) The muscular coat was filled with large blood spaces, giving the organ the appearance of erectile spongy tissue. (iv) The uterine body rather than the cervix underwent alteration and growth as puberty approached.

Urological Exhibits.

DR. R. BRIDGE, DR. K. KIRKLAND and DR. A. C. TELFER showed some specimens and corresponding pyelograms. The first case discussed was that of a woman, aged fifty-three years, who had been subjected to nephrectomy for a papillary carcinoma involving the middle calyx. One year later she returned with papillomatosis of the bladder, which had spread from a small portion of the ureter left at operation. The remaining part of the ureter was removed and the bladder papillomata were destroyed by diathermy. Except for two small recurrences, which were destroyed, the patient had been clear of signs and symptoms for the past six months. Dr. Bridge emphasized the importance of total ureterectomy in dealing with papillary carcinoma, even when at the primary operation recurrence seemed most unlikely. He had in this case at the second operation passed a diathermy electrode into the intramural portion of the ureter and had thoroughly destroyed the mucous membrane there. Dr. Bridge also stressed the importance of frequent post-operative inspection for papillary recurrence in the bladder.

A case of retentive diverticulum of the bladder, with successful treatment by operation, was revealed in a series of cystograms taken before and after operation.

The difficulties of operating on a solitary kidney were shown in two cases. The first was that of a man, aged twenty-nine years, who had been subjected to left nephrectomy for injury nine years previously, and who had a stone in the remaining kidney. The second was that of a man, aged forty-three years, who had very poor function in a calculous pyonephrotic kidney on the left side and a stone at the uretero-pelvic junction on the right side, on which the kidney was functioning moderately well. In the first case, the function of the only kidney being good, nephrolithotomy had been performed. In the second case nephrolithotomy and nephrostomy were performed on

the left or worse side first, in an endeavour to restore as much function as possible. After the operation examination following injection of "Uroselectan" revealed little restoration of function; operation was performed on the better side, and the uretero-pelvic stone was removed. Dr. Kirkland, in discussing this case, pointed out the modern trend towards more conservative renal surgery. Formerly it was said that coccal infection of a calculous kidney meant nephrectomy; but with the advent of efficient antiseptic therapy the tendency was to conserve this kidney, although the problem of the solitary kidney would always be a source of worry.

It was stated that the routine method adopted at Sydney Hospital for obtaining increased information from excretion urograms, especially in suggested pelvi-ureteral kinking, was to take films in inspiration and expiration, and in the Trendelenburg and reverse Trendelenburg positions. This was illustrated in a series of X ray films of a girl, aged twenty-eight years, who had complained of severe intermittent right renal pain, and whose urograms revealed a constant kinking just below the uretero-pelvic junction. She had been relieved by the freeing of a band of adhesions at the demonstrated level on the ureter.

A series of pyelograms made pre-operatively and post-operatively in a case of right renal proptosis, the kidney being at the pelvic brim, were shown. Dr. Bridge explained that at operation he had performed nephropexy, a Rammstedt's operation on the narrow pelvic outlet, and renal sympathectomy. The films taken after operation showed a greatly reduced pelvis and a diminution of the calyceal dilatation. Dr. Telfer said that Dr. Hinman, of San Francisco, laid particular stress on the importance of renal sympathectomy for the relief of pain in renal proptosis.

NOMINATIONS AND ELECTIONS.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Barron, Andrew Moncrieff, M.B., B.S., 1938 (Univ. Sydney), Esplanade, Manly.

Lawrie, Charles Malcolm, M.B., B.S., 1927 (Univ. Sydney), Prospero Street, Murwillumbah.

McEntire, Ronald Gordon John, M.B., B.Ch., 1912 (Dublin), B.A.O., 225, Macquarie Street, Sydney.

Post-Graduate Work.

MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

THE Melbourne Permanent Post-Graduate Committee has arranged courses of clinical demonstrations in preparation for the second parts of the examinations for the degrees of Doctor of Medicine and Master of Surgery. The programmes are as set out below.

Doctor of Medicine.

Diseases of the Urinary System.—Dr. W. W. S. Johnston will give demonstrations of diseases of the urinary system at 4.30 p.m. each Monday from May 2, 1938, to May 23, 1938.

Diseases of the Cardio-Vascular System.—Dr. H. H. Turnbull will give demonstrations of diseases of the cardio-vascular system on Wednesday, May 4, 1938, at 4.30 p.m.; Saturday, May 7, at 11.15 a.m.; Wednesday, May 11, at 4.30 p.m.; and Saturday, May 14, at 11.15 a.m.

Diseases of the Nervous System.—Dr. R. P. McMeekin will give demonstrations of diseases of the nervous system at 4.30 p.m. on Wednesday, April 18; 11.15 a.m. on Saturday, May 21; 4.30 p.m. on Wednesday, May 25; and 11.15 a.m. on Saturday, May 28, 1938.

Dr. E. G. Robertson will give demonstrations of diseases of the nervous system at 4.30 p.m. on Wednesday, July 13; 11.15 a.m. on Saturday, July 16; 4.30 p.m. on Wednesday, July 20; and 11.15 a.m. on Saturday, July 23.

Diseases of the Alimentary System.—Dr. F. Blois Lawton will give demonstrations of diseases of the alimentary system at 4.30 p.m. on Wednesday, June 1; 11.15 a.m. on Saturday, June 4; 4.30 p.m. on Wednesday, June 8; and 11.15 a.m. on Saturday, June 11, 1938.

Diseases of the Blood and Lymphatic System.—Dr. K. D. Fairley will give demonstrations of diseases of the blood and the lymphatic system on Wednesday, June 15, 1938, at 4.30 p.m.; Saturday, June 18, at 11.15 a.m.; Wednesday, June 22, at 4.30 p.m.; and Saturday, June 25, at 11.15 a.m.

Diseases of the Respiratory System.—Dr. L. E. Hurley will demonstrate diseases of the respiratory system at 4.30 p.m. on Wednesday, June 29; 11.15 a.m. on Saturday, July 2; 4.30 p.m. on Wednesday, July 6; and 11.15 a.m. on Saturday, July 9, 1938.

Modern Methods of Investigation.—Dr. Douglas Thomas will demonstrate modern methods of investigation on Wednesday, July 27, 1938, at 4.30 p.m., and Saturday, July 30, 1938, at 11.15 a.m.

The demonstrations will be, for the most part, of a clinical nature. The systemic subdivision will not necessarily be strictly followed. Interesting patients available at the time may be substituted for demonstration. The demonstrations will be held in the wards, the class room or the lecture theatre of the Walter and Eliza Hall Institute, Royal Melbourne Hospital.

The fee for this course will be ten guineas.

Psychiatry.—During May, June and July, Dr. J. K. Adey will give a series of weekly demonstrations in psychiatry, at a time arranged by mutual agreement, at the Receiving House, Royal Park.

The fee for this course will be three guineas.

Master of Surgery.

Twenty-four clinical demonstrations will be given during the months of May, June and July in the X ray lecture room of the Alfred Hospital, Melbourne, on Wednesdays at 4.15 p.m. and on Saturdays at 11.15 a.m.

The following six members of the staff of the Alfred Hospital will each conduct four demonstrations: Dr. Fay Maclure, Dr. J. Kennedy, Dr. A. J. Trinca, Dr. L. H. Ball, Dr. C. J. O. Brown, Dr. C. A. M. Renou.

The fee for this course will be ten guineas.

A course in surgical pathology is being conducted coincidently at the Department of Pathology within the Melbourne University. It has been decided to allow a limited number of medical practitioners to attend these demonstrations, in addition to the candidates for examination, on the understanding that preference in regard to examination of patients will be reserved for the candidates.

Correspondence.

NATIONAL HEALTH INSURANCE.

SIR: In *The Sydney Morning Herald* of the fourteenth instant there appear preliminary details of the scale of remuneration for doctors employed under the national insurance scheme. The terms, providing for an annual payment of 11s. in respect of each insured person, seem satisfactory, and while covering all those services which may reasonably be expected of a general practitioner, rightly exclude "treatment in respect of a confinement, treatment covered by workers' compensation acts, major operations, and the administration of an inhalant anæsthetic". In passing, the use of the plural would be preferable in the first and last items, just as it is in the third.

But why specify inhalant anæsthesia? Are not other methods not only possible, but often obligatory? Shall

insured patients be deprived of the comforts and benefits of local and intravenous anæsthesia simply because the doctor's source of profit must be guarded? This assuredly will be a consideration when the choice of anæsthetic is under review in such cases. Will not indications for intravenous, regional or spinal anæsthesia often arise, and shall ether be clapped on notwithstanding? Gas certainly will be avoided owing to the cost of apparatus and material.

Perhaps those who have formulated these conditions have worked better than we know: they may be so enlightened as to have recognized that such alternative methods demand a certain amount of specialized knowledge and skill, and that therefore they are beyond the scope of the average general practitioner. Perhaps they are making provision for the application of such methods by specialists only—a desirable but almost Utopian objective, and one certainly not reconcilable with the conditions of medical practice generally obtaining in this country.

In practice, of course, the situation will be that everyone, except perhaps the moribund, will get ether. It will be given to cases of incipient and even active tuberculosis. For greater economy and larger margin of profit patients will be bludgeoned into unconsciousness by that wicked and barbarous method, closed ether. Surely no encouragement should be given to the subordination of the patient's comfort, safety and future welfare, to considerations of financial expediency, as will be possible if this restriction remains unchallenged. The present custom of regarding the anæsthetic as the prerogative of the referring doctor, largely irrespective of his qualifications or of the merits of the case, is bad enough, and anything which will further hamper the rescue of anæsthesia from the comparative neglect and degradation which it now suffers is to be strongly resisted.

I submit, sir, that the provision excluding inhalant and presumably including all other forms of anæsthesia as benefits is in the interests neither of the profession in general and specialists in particular, nor of the insured patient. Steps should be at once taken by those responsible to have the word "inhalant" deleted, in its relation to anæsthesia, from the conditions of service now being laid down.

Yours, etc.,

S. V. MARSHALL.

135, Macquarie Street,
Sydney,
April 15, 1938.

MATERNITY AND SOME OF ITS PROBLEMS.

SIR: I wish to take the opportunity of congratulating Dr. McLelland on his address as given in the journal of the sixteenth instant. His analysis of the natural relation of the family unit is lucid, virile and full of truth. But I cannot agree with his view of the intrinsic degeneracy of modern men.

The fact that the patriarch has been replaced by the pensioner is not the fault of the men of today; unless it be that politicians, at the behest of financiers and industrialists, have allowed the Frankenstein of the modern economic system to develop. The lower middle-class and basic-wage men do the best, in most cases, that circumstances will allow. Dr. McLelland himself could not worthily uphold the rôle of patriarch on the basic wage, or thereabouts, with the spectres of depressions and unemployment forever at his door.

In close on a quarter of a century of suburban general practice I have formed the opinion that paternal affection still lives robustly under circumstances which make it little short of heroic. The fact that approximately 80% of men practice the functional mutilation of *coitus interruptus* is proof positive of their consideration and restraint. And I find that when, accidentally, an unwanted pregnancy does occur, both husband and wife, in the vast majority of instances, face up to it, and even show a joy when the child arrives which is both noble and pathetic. The people are all right; the system which thwarts their natural instincts and condemns them to poverty and their offspring to insecurity is solely to blame.

Yours, etc.,

HENRY HALLORAN.

Norwood,
South Australia,
April 21, 1938.

PULMONARY TUBERCULOSIS.

SIR: In reviewing Dr. Wingfield's book on pulmonary tuberculosis, in the journal of April 23, your reviewer disagrees with Wingfield's suggestion that a pleural effusion should be replaced only if there is evidence of lung involvement, and states that the only safe rule is to replace every effusion of tuberculous origin. I should be interested to know how, in the absence of evidence of lung involvement, your reviewer proposes to determine whether any given effusion is of tuberculous origin.

Yours, etc.,

H. M. L. MURRAY.

Department of Defence,
Maribyrnong,
Victoria.
April 29, 1938.

Analytical Department.

LUPI "GOLD MEDAL" OLIVE OIL.

"GOLD MEDAL" OLIVE OIL is manufactured by Joseph Lupi, Porto Maurizio, Italy, and is distributed in this country by John Vose, Proprietary, Limited. It is stated to be a pure olive oil, suitable for medical use. A sample was submitted to our analysts, who report as follows:

We have examined the sample of olive oil submitted by you and found as follows:

		Required by British Pharmacopœia (1932).
Specific gravity at 15·5° C. ..	0·9166	0·915-0·918
Refractive index at 40° C. ..	1·4615	1·4605-1·4635
Acid value	1·12	Not more than 2·0
Saponification value	190·4	190-195
Iodine value	81·8	79-88

British Pharmacopœia test for presence of—

Cottonseed oil: negative.
Sesame oil: negative.
Arachis oil: negative.

The sample complies in all respects with the requirements of the British Pharmacopœia (1932).

Medical practitioners might prescribe Lupi "Gold Medal" olive oil with confidence.

Proceedings of the Australian Medical Boards.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1925 *to* 1935, of Queensland, as duly qualified medical practitioners:

Adam, Geoffrey William Shedden, M.B., B.S., 1933 (Univ. Sydney), Brisbane.
Kerr, Edith Margaret, M.B., B.S., 1937 (Univ. Melbourne), Brisbane.
Miller, Elizabeth Constance Graham, M.B., B.S., 1937 (Univ. Melbourne), Townsville.
Muller, Joy Helena, M.B., B.S., 1938 (Univ. Melbourne), Brisbane.
Ross, Angela Mary, M.B., 1928 (Univ. Sydney), Mackay.
Shaw, Francis William Rutherford Seymour, M.B., 1932 (Univ. Sydney), Jundah.

SOUTH AUSTRALIA.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Practitioners Act of* 1919, of South Australia, as duly qualified medical practitioners:

Benson, Henry Gordon, M.B., B.S., 1936 (Univ. Sydney), Northfield Infectious Diseases Hospital, Northfield.

Phillips, Frank, M.R.C.S. (England), L.R.C.P. (London), 1928, Berri.

Donnelly, Thomas Hewson, M.B., B.S., 1937 (Univ. Adelaide), Adelaide Hospital, Adelaide.

Nankivell, Elizabeth, M.B., B.S., 1937 (Univ. Melbourne), North Adelaide.

Salts, Richard John, M.B., B.S., 1935 (Univ. Melbourne), Millicent.

Hopkins, Charles McTaggart, M.B., B.S., 1934 (Univ. Melbourne), Mount Lofty.

Books Received.

NOTES ON BACTERIOLOGY AND CLINICAL PATHOLOGY FOR NURSES, by H. Rogers, M.D., Ch.B.; 1938. London: H. K. Lewis and Company Limited. Crown 8vo, pp. 40. Price: 1s. net.

IDEAL WEIGHT: A PRACTICAL HANDBOOK FOR PATIENTS, by W. F. Christie, M.D.; 1938. London: William Heinemann (Medical Books) Limited. Crown 8vo, pp. 121. Price: 5s. net.

POULSSON'S TEXT-BOOK OF PHARMACOLOGY AND THERAPEUTICS; Second English Edition, thoroughly revised by S. Alstead, M.D., M.R.C.P.; 1938. London: William Heinemann (Medical Books) Limited. Royal 8vo, pp. 568. Price: 25s. net.

FRACTURES AND DISLOCATIONS FOR PRACTITIONERS, by E. O. Geckeler, M.D.; 1937. London: Baillière, Tindall and Cox. Medium 8vo, pp. 262, with illustrations. Price: 25s. net.

EMERGENCY SURGERY, by H. Bailey, F.R.C.S.; Third Edition; 1938. Bristol: John Wright and Sons Limited; London: Simpkin Marshall Limited. Medium 8vo, pp. 852, with 816 illustrations, of which many are in colour. Price: 50s. net.

Diary for the Month.

MAY 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MAY 13.—Queensland Branch, B.M.A.: Council.
MAY 17.—New South Wales Branch, B.M.A.: Ethics Committee.
MAY 18.—Western Australian Branch, B.M.A.: Branch.
MAY 19.—New South Wales Branch, B.M.A.: Clinical Meeting.
MAY 24.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAY 25.—Victorian Branch, B.M.A.: Council.
MAY 26.—New South Wales Branch, B.M.A.: Branch.
MAY 26.—South Australian Branch, B.M.A.: Branch.
MAY 27.—Queensland Branch, B.M.A.: Council.
JUNE 1.—Victorian Branch, B.M.A.: Branch.
JUNE 1.—Western Australian Branch, B.M.A.: Council.
JUNE 2.—South Australian Branch, B.M.A.: Council.
JUNE 3.—Queensland Branch, B.M.A.: Branch.
JUNE 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii-xx.

FREMANTLE HOSPITAL, FREMANTLE, WESTERN AUSTRALIA: Medical Superintendent.
GOVERNMENT OF NORTH BORNEO: Medical Officer.
ROYAL HOSPITAL FOR WOMEN, PADDINGTON, NEW SOUTH WALES: Resident Medical Officers.
SYDNEY HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Clinical Assistant.
TAMBO HOSPITALS BOARD, TAMBO, QUEENSLAND: Medical Superintendent.
THE LORD HOWE ISLAND BOARD OF CONTROL: Medical Officer.
WOMEN'S HOSPITAL, MELBOURNE, VICTORIA: Pathologist.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

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